**PART I**

# ANTI ULCER ACTIVITY OF *"PANAI POO CHOORANAM"* (*Borassus flabellifer Linn*)

**&**

**PART II**

# HEPATOPROTECTIVE ACTIVITY OF *"ARITHIRAADHI CHOORANAM"*

The dissertation Submitted by

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*Under the Guidance of*

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## GOVT. SIDDHA MEDICAL COLLEGE,

## CHENNAI-106

## DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"Anti-Ulcer Activity of *Panai poo chooranam (Borassus flabellifer, Linn)"* and "Hepatoprotective Activity of *Arithiraadhi chooranam*"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. A. Kumar MD (Siddha)**, HOD, Post Graduate Department of Gunapadam, Govt.Siddha Medical College, Arumbakkam, Chennai-106 and the dissertation has not formed the basis for the award of any Degree, Diploma, Fellowship or other similar title.

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## ENDORSEMENT BY THE HOD,

## PRINCIPAL/HEAD OFTHE INSTITUTION

This is to certify that the dissertation entitled **"Anti ulcer Activity of *Panai poo chooranam (Ehretia microphylla, Lamk)"* and "Hepatoprotective Activity of *Arithiraadhi chooranam*"** is a bonafide work carried out by Dr.P.Kavitha under the guidance of **Dr. A. Kumar, M.D (Siddha)**, HOD, Post graduate department of Gunapadam, Govt.Siddha Medical College, Chennai - 106.

**Seal and Signature of the HOD** **Seal and Signature of the Principal**

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**Place:** Chennai

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# CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"Anti – Ulcer Activity of *Panai poo Chooranam (Borassus flbellifer* Linn*)*** and **"Hepatoprotective Activity of *Arithiraadhi chooranam "*** is submitted to the Tamilnadu Dr.M.G.R.Medical University in partial fulfillment of the requirements for the award of degree of M.D (Siddha) is the bonafide and genuine research work done by **P.Kavitha** Under my supervision and guidance and the dissertation has not formed the basis for the award of any Degree, Diploma, Associateship, Fellowship or other similar title.

**Date:** **Seal & Signature of the Guide**

**Place:** Chennai

**Abbrevations:**

| | | |
|---|---|---|
| AC | - | Air chambers |
| AIDS | - | Acquired Immuno Deficiency Syndrome |
| Alb | - | Albumin |
| ALP | - | Alanine Phosphatase |
| ALT | - | Alanine Transaminase |
| ANOVA | - | Analysis of Variance |
| AST | - | Aspartate Transaminase |
| AT | - | After Treatment |
| BCF | - | Bundle Cap Fiber |
| BT | - | Before Treatment |
| $CCl_4$ | - | Carbon Tetra Chloride |
| Cl | - | Cholesterol |
| CMC | - | |
| CNS | - | Central Nervous System |
| COX | - | Cyclooxygenase |
| DC | - | Differential Count |
| Dep | - | Deposits |
| DU | - | Duodenal Ulcer |
| E | - | Eosinophil |
| EP | - | Epidermis |
| ESR | - | Erythrocyte Sedimentation Rate |
| FB | - | Fiber Bundle |
| FPC | - | Few Pus cells seen |
| FTIR | - | Fourier Transformer Infrared Spectroscopy |
| GIT | - | Gastro Intestinal Tract |

| | | |
|---|---|---|
| GP | - | Ground Parenchyma |
| GT | - | Ground tissue |
| Hb | - | Hemoglobin |
| IEC | - | Institutional Ethical committee |
| IRB | - | |
| L | - | Lymphocyte |
| LFT | - | Liver Function Test |
| NSAID | - | Non Steroidal Anti Inflammatory Drugs |
| OE | - | Outer Epidermis |
| OECD | - | Organization for Economic Corporation and Development |
| P | - | Polymorphs |
| PCS | - | Pus Cells Seen |
| Ph | - | Phloem |
| PPT | - | Precipitate |
| PUD | - | Peptic ulcer disease |
| RFT | - | Renal function Test |
| Sc | - | Sclerenchyma |
| SEM | - | Scanning Electron Microscope |
| St | - | Stomata |
| TB | - | Tuberculosis |
| TC | - | Total Count |
| TLC | - | Thin Layer Chromatography |
| VB | - | Vascular bundle |
| VS | - | Vascular Strand |
| WHO | - | World Health organization |
| X | - | Xylem |

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# INTRODUCTION

Siddha is a system of medicine which catered the health needs to the people. This scientific system was originated by the spiritual scientists, they are known as Siddhars. Siddha System is mostly therapeutic in nature and its origin can be traced back to the birth of human race on the planet.

Siddha defines health as a perfect state of physical, psychological, social and spiritual wellbeing of an individual. Siddha therapy recognizes the importance of body, mind and vital energies. Siddha treatment is essentially aimed at restoring balance to the individual's system by addressing all these aspects. It is a unique method which brings a person to a holistic state of existence rather than just being free from disease.

In siddha literature *AgathiyarRathinaSurukam* classified 4448 diseases. In this classification the disease *Gunmam* was described.

The disease *Gunmam* is correlated with the modern terminology peptic ulcer disease.

Peptic ulcer disease comprises ulcers in the stomach or duodenum.

Ulceration of the gastro intestinal tract due to the combined action of hydrochloric acid and pepsin. This occurs most commonly in either the stomach or duodenum but it may also occur in the oesophagus, Meckel's diverticulum, jejunum.

Imbalance between the damaging effects of acid and pepsin attack and the body's mucosal diffences.

Psychological factors – Stress of modern life is often invoked as an important cause.

A major causative factor (60% of gastric and up to 90% of duodenal ulcers) is chronic inflammation due to ***Helicobacter pylori*** that colonizes the antral mucosa.

Other important contributing factors are NSAIDs.

Additional aggressive factors include smoking, ethanol, bile acids, aspirin, and steroids.

PUD are very common in India, with 4 Million individuals affected per year, Life time prevalence of PUD in the India is approximately 12% in men and 10% in women. More over an estimated 15,000 deaths per year occur as a consequence of complicated PUD. (Agreus and Tally, 1997).

In India the prevalence of peptic ulcer is quite high. 4 to 10 thousand populations suffers from peptic ulcer disease every year. Tamil Nadu, Karnataka, Andhra Pradesh and Jammu & Kashmir are considered to be very high risk area. ( WHO 2011 )

Peptic ulcer disease had a tremendous effect on morbidity and mortality until the last decades of the 20th century. According to the latest WHO data published in April 2011 Peptic Ulcer Disease Deaths in India reached 108,392 or 1.20% of total deaths. The age adjusted Death Rate is 12.37 per 100,000 of population ranks India is 5th in the world.

Current treatment of ulcers in developing countries has been largely suppression of pain, with little or no strategy aimed at a cure. Although a number of antiulcer drugs such as $H_2$ receptor antagonists and proton pump inhibitors are available for ulceration all these drugs have adverse reactions such as hypersensitivity, arrhythmia, impotence and haemopoietic changes with is a possibility of increased rate of ulcer recurrence within one year after cessation of the treatment. (Ariyoshi *et al.*)

As society continue to change with the approach of new century the caring values of nature remains the hallmark of its unique contribution to health. Anticipating and planning to live provide man a great challenge without plants.

Plants being the natural medicines we are bound to live with nature to be free from diseases. Siddha system of medicine attacks the very root cause of the disease, there by curing the diseases completely without any adverse effects.

Nature is the material cause not merely the outer view but also of our body. So man is the mini representation of the universe.

Herbal medicine is fast emerging as an alternative treatment to available synthetic drugs for treatment of ulcer possibly due to lower costs, availability, fewer adverse effects and perceived effectiveness. The World Health Organization (WHO) estimates that 80 percent of the populations of some Asian countries presently use herbal medicine for their illness.

A Short History of Medicine

2000 B.C. - "Here, eat this root."

1000 B.C. - "That root is heathen, say this prayer."

1850 A.D. - "That prayer is superstition, drink this potion."

1940 A.D. - "That potion is snake oil, swallow this pill."

1985 A.D. - "That pill is ineffective, take this antibiotic."

2000 A.D. - "That antibiotic is artificial. Here, eat this root."

So this world starts realizing the importance of herbs and it is the right time to show the significance of *siddha* system.

Also it is our primary duty to establish Siddha medicines with scientific datas.

The herbal drug ***panai poo (Borassus flabellifer)*** is easily available in our native place.

So I decided to treat the Peptic ulcer disease with the herbal medicine ***Panai poo (Borassus flabellifer)***.

# 2. AIM AND OBJECETIVES

## Aim

Various herbal plants were used therapeutically in the treatment of Peptic ulcer disease. In our Siddha text book, flower of *Borassus flabellifer* is mentioned for treating peptic ulcer. Till now *Panai poo chooraanam* has not been scientifically evaluated for treating peptic ulcer. The principle aim of this study is to evaluate the efficacy of the drug ***Panai poo Chooranam*** in the management of Peptic ulcer disease in pre- clinical and clinical aspects.

## Objectives:

In this dissertation work, the "***PANAI POO CHOORANAM***" is analyzed to assess the following aspects:

- Identification of the herbal drug *Panai poo*
- To get the authentication of the raw drug
- To collect the literature review
- Pharmacognostic study for the raw drug
- Phytochemical and Chemical analysis for the trial drug to identify the active components
- Evaluation of the toxicity of test drug
- Pharmacological study to evaluate the anti – ulcer activity
- Clinical assessment of the *Panai poo chooranam.*

# 3. REVIEW OF LITERATURE

## 3.1 BOTANICAL ASPECT OF *Borassus flabellifer*

**Botanical name:** *Borassus flabellifer Linn*

**Scientific classification-** ( Bentham and Hooker)

| | | |
|---|---|---|
| Kingdom | - | Plantae |
| Division | - | Magnoliophyta |
| Class | - | Monocotyledons |
| Subclass | - | Arecidae |
| Order | - | Arecales |
| Family | - | Arecaceae |
| Genus | - | *Borassus* Linn |
| Species | - | *Borassus flabellifer* Linn |

**Vernacular names**

| | | |
|---|---|---|
| Eng | - | Palmyra palm, brab tree |
| Tamil | - | *Thaalam, Karumpuram, Edagam, Kaamam, Tharuviragan, Thaali* |
| Sans | - | *Tala* |
| Hin | - | *Tar* |
| Tel | - | *Tati* |
| Mal | - | *Pana* |
| Kan | - | *Pane-amra* |
| Beng | - | *Tal* |
| Guj | - | *Tad* |
| Oriya | - | *Talo* |
| Assam | - | *Tal* |

**Description of the plant**

The palmyra tree is the official tree of Tamil Nadu. Highly respected in Tamil culture, it is called "*karpaha Viruksham*" ("celestial tree") because all its parts without exception have a use.

*Borassus* is derived from a Greek word describing the leathery covering of the fruit and *flabellifer* means “fan-bearer”.

*Borrasus flabellifer* is a very tall, erect, magnificent dioecious palm, 20-30 m in height and 1.0-2.2m in girth, with a fine crown of 30-40 large leaves, found throughout tropical India, especially along the peninsular coast and in West Bengal and Bihar.

Trunk blackish grey, cylindric, with a dense mass of long rootlets near the ground, generally straight, swollen above the middle and again contracting upwards, old stems marked with black, narrow scars of petioles, young stems covered with dry leaves or with the bases of their petioles, leaves palmately divided, fan shaped, petioles 0.6-1.2m long, stout, semi terete, spines cent-margined, lamina 0.9-1.5m in diam, rigidly coriaceous, divided into lanceolate or linear 2-fid lobes, segments 60-80, shining, folded along the midrib, spinulose; spadices very large, stout, male spadix stout, cylindric, branched, or sometimes double, bracts enclosing spikelets, flowers yellow; female spadix sparingly branched, flowers yellow, solitary, few scattered; drupes 15-20cm in diam, enclosed by the enlarged perianth, distinctly trigonous when young, almost spherical when old; pyrenes 3-1, obcordate, fibrous outside with hyaline edible endosperm.

Most tapped palm trees do not only produce sap but are multipurpose (edible fruits, building materials, fuel, fibres, wax, etc.) and their socio-economic importance can be critical for the rural poor.

**Father of our nation mahathma Gandhiji used to call *Borassus flabellifer*, as a remedy against poverty.**

The palmyra probably native to India, is the most striking of the palms and is a grand feature in the landscape of the tropical regions. It has run wild in many parts of India and has also been cultivated, chiefly in the dry or sandy localities of Andhra Pradesh, Bihar, Karnataka, Kerala and Madhya Pradesh, Orissa, Tamil Nadu and West Bengal. Several large groves occur in some of these areas. It grows in isolated patches in other states such as Assam, Gujarat, Maharashtra and Uttar Pradesh. The total palm is estimated at 76,167,000 trees, and the tappable trees at 56,998,00. It is found in the plains along the riverbanks and coastal areas, and grows on every type of wasteland, including rocky areas. The most congenital situations for its favorable development are low sandy plains, scarcely above the sea-level, where they are exposed to the burning sun and at least one monsoon. It is distinctly wild and propagates itself readily from seed in regions where it is abundant. In such regions, it is capable of forming pure forests. The palmyra sometimes acts as a windbreak for the plains.

**Cultivation**

The palm grows naturally, and no particular cultivation is necessary.

It requires no artificial irrigation or manuring. It is generally propagated by direct sowing. Poly embryony and twin seedlings have been reported in the palm, although in general only one seedling emerges.

It flowers during March-May in some areas and produces the ripe fruits during Aug-Sep, whereas in some other areas flowering is during Nov-Feb.

**Chemical Constituents**

Gum, fats, albuminoids, steroidal glycosides and carbohydrates like sucrose.

**Utilization**

Every part of the palm is useful. It is a source of food, a sweet drink (sweet toddy), jaggery, sugar, vinegar, palm-wine, medicine and wood.

The leaves are used for thatching, umbrellas, fans, diaper articles, hats, and their fibres for baskets, brushes and brooms.

The fresh saccharin juice obtained by excision of the spadis early in the morning is cooling, Stimulant and antiphlogist, also acts as a laxative taken regularly for several mornings. It is useful for inflammatory affections and dropsy. It is also useful in gonorrhea.

Sweet toddy- the spadices of palmyra, on tapping, yield a delicious sugary sap, known as the sweet toddy.

The fresh sap is a cheap, refreshing and delicious beverage, with an agreeable flavor. It is a nutricious supplement to diets which are deficient in iron, ascorbic acid, and vitamin-B complex.

It is cooling, diuretic, stimulant, antiphlegmatic, laxative, and is also useful in inflammatory affections, ulcers, and dropsy. Slightly fermented juice is given in diabetes.

The sap is given as a tonic to asthmatic and anaemic patients, and in Hansen's disease. It is an excellent source of biologically available riboflavin.

The sap is an excellent source of vinegar of very fine quality and of very attractive white colour.

The toddy poultice prepared by adding fresh drawn toddy to rice flour and subjected to a gentle fire till fermentation takes place, then spread on a cloth forms a valuable stimulant application to gangrenous and intolent ulcers, carbuncles.

The terminal buds of the tree are regarded as nutritive, diuretic and tonic.

**Figure No: 3.1 FLOWER OF *BORASSUS FLABELLIFER***

The ashes of the flowering stalk are set to the useful in enlarged spleen.

The decoction of the bark with a little salt added to it is a good astringent gargle for strengthening gums and teeth.

The palm jaggery is prepared by boiling the sweet juice. The jaggery is dark, with a characteristic flavor. Carbohydrates are the principal constituents of palm-jaggery. It contains of vitamins of B group and minerals. The concentration of amino acids in palm-jaggery is much high. It exhibits mild laxative action and is reported to be an effective therapeutic agent for anaemia.

The sweet palm candy is directly produced from the sap. It is considered a delicacy. It is reputed to possess medicinal properties, and is used in coughs and pulmonary affections, and as laxative for children.

The milky fluid from the immature seeds is sweet and cooling and prevents hiccup and sickness.

The root is considered cooling, restorative, diuretic, and anthelmintic. It cures gonorrhoea.

Ash of the spathe is useful as an antacid in heart-burn and as an antiperiodic.

Extract of green leaves given in secondary syphilis.

The flower and root are reported to be used in Kampuchea as sinapism in tumours of uterus. Gum act as emollient in indurations.

[Ref: The Wealth of India, The Indian materia medica, Indian plants and drugs with their medical properties and uses, Compendium of Indian medicinal plants ]

## 3.2 GUNAPADAM ASPECT OF THE PLANT

***Panai- Borassus flabellifer***

**Other tamil names:**

*Thaalam, Karumpuram, Edagam, Kaamam, Tharuviragan, Thaali*

**Useful parts:**

*Kuruthu*, leaves, flower, *nongu*, fruit, *mattai*, root tuber.

**Leaf, *mattai*, root**

| | | |
|---|---|---|
| Taste | - | Astringent, sweet |
| *Thanmai*(Character) | - | *Thatpam* |
| *Pirivu*(classification) | - | Sweet |

**Flower**

| | | |
|---|---|---|
| Taste | - | Astringent |
| *Thanmai*(Character) | - | *Thatpam* |
| Pirivu(Classification) | - | Sweet |

**Toddy**

| | | |
|---|---|---|
| Taste | - | Sweet; |
| *Thanmai*(Character) | - | *Thatpam*; |
| *Pirivu*(Classification) | - | Sweet |

**Actions:**

**Leaf, *mattai*** : Astringent
Aphrodisiac

**Tender palm fruit**: Diuretic
Demulcent
Nutrient

**Sweet toddy:** Diuretic
Refrigerant

**Toddy:** Stimulant
Antiphlogistic

**General characters of *Kuruthu*:**

*Kuruthu* stimulates conditions like bleeding piles and diarrhoea.

**General characters of flower:**

"□□□□□□□□□□ □□□□□□□□□□ □□□□□□□□□□□

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□□□□□□□ □□□□□ □□□□□□□□". அகத்தியர் குணவாகடம்

பனை பூவினால் வாத குன்மம், நீரருகல், பல் நோய், பழஞ்சுரம் குணமாகும்.

**Medicinal uses**

The tender *nongu* grown in palm trees of *neithal nilam* is indicated for *viyarvai kuru*. It stimulates hunger. *Nongu* along with its covering cures dysentery.

If palm fruit consumed as food everyday produces eczema, scabies, constipation diseases related to pitha vayu.

Dry leaf used as hard fan will cure *vatha, pitha, kapa* diseases and aguesia.

Toddy develops aphrodisiac action and stamina.It purifies all types of *pashanas*.

Sweet candy (*panamkarkandu*) relieves *megasuram*, heat due to small pox, *neerchurukku* and thirst.

Jaggery cures diseases emerged due to *tridoshas*, delirium, aguesia and ulcer.

**Jaggery decoction**

Jaggery and dry ginger are made as decoction and if taken during morning relieves fever, indigestion, venereal disease and dropsy.

Male flower is burnt and mixed with water discarding the sediment, the water if heated produces salt. Process is repeated for 3 times and end salt is given in small amount has diuretic action. It cures splenomegaly and hepatomegaly.

Juice of palm *mattai* is used in the treatment of eye diseases.

Tender *kuruthu* is edible. It is sweeter but it has laxative action.

This tree is compared to Beema. Beema is compared to mercury. So the decoction of root tuber is used in the calcification process of mercury (*Rasa Parpam*)

Sweet toddy or *patha neer* consumed without any interval for 40 days cures *meganoi*.

Toddy is used in purification of *pashanas.*

Ash of the floweris used externally for wounds. If given internally has diuretic and laxative action.

The unriped fruit has coolant action. It is used during summer to relieve thirst.

The ripped fruit has hard nut and outer fleshy part. It produces indigestion. The poor people used to consume this fruit as burnt or boiled form.

This root tuber gives strength. Dried root tuber ground to flour is mixed with coconut milk and salt is boiled and taken regularly gives strength. In the boiled root tuber the skin is removed and made into pieces and dried. The flour is used in the preparation of snacks give strength and weight gain to the body.

The pieces of root with two parts of water kept in *suriyapudam* then heated in *deepakini* then the water is collected and taken regularly during morning, noon and evening.

Ref: Siddha Materia Medica (Medicinal Plants Division)

**Other Preparations of *Borassus flabellifer* :**

***Keezhanelli girutham***

Root of *Phyllanthus amarus* - 3 parts

Root of *Bambusa arundinaceae*
Root of *Amaranthus tricolor*
**Root of *Borrasus flabellifer***
Root of *Pterocarpus marsupium*
*Keeda ver* directed towards east side
} Each 1 part

All above roots are dried well, and taken in a pot add 8 parts of milk and add equal parts of water, properly boiled and decoction is prepared.

To the decoction add,

Ficus *racemosa, Terminalia chebula, Terminalia bellerica, Phyllanthus emblica*, seeds of greens, cardamomum, Ficus *benghalensis*, sugar, ghee are heated and taken in a good consistency.

Therapeutic indications:

- Joint disorders
- Epilepsy
- Heat diseases in the lower abdomen
- Blackens the hair as that of seeds of green, and gives beauty.

*Theraiyar maga karisal*, Pg no: 70

***Neerkatuku parigaaram***

*Achyranthus aspera*

**Flower of *Borassus flabellifer***

*Vazhai sarugu*

All the above should be dried properly, and each ingredients should be made burnt and made into ash separately. Then equal parts of the ash about 70gms is mixed with,

| | | |
|---|---|---|
| Purified | Alum | each 35gms |
| " | Sodium chloride impure | |
| " | Fullers earth | |
| " | Borax | |

All ingredients mixed well, and kept in a air tight container.

Dose : 2-3 *kunri* ; twice daily

Adjuvent : honey

Therapeutic indications: Renal calculi

*Kannusamy parambarai vaithiyam* pg no: 462

**Decoction for *vayu gunmam:***

**Dried flower of *borassus flabellifer***

Whole plant of *plambago zeylanica*

Whole plant of *Achyranthus aspera*

Each one is burnt separately, and white ash is prepared, mixed in equal proportion.

Take 35gm of above said ash and mixed with 3.9 lrs of water, stirred well kept throughout the night and filtered in the morning. Above filtrate is taken, added with horse gram and then it is allowed to boil till it becomes 1 part. Then the reduced filtrate is again filtered, added with honey and *thirikadugu* powder.

*Theraiyar vagadam*, pg no: 162

**Root of Borassus *flabellifer*** is one of the ingredients of following medicines.

*Bosana gudori nei* - *Theraiyar maga karisal*, pg no: 169

*Peru viyadhiku nei* - *Theraiyar maga karisal*, pg no: 163

*Koovilai legiyam* - *Theraiyar maga karisal*, pg no: 74

*Uthamani kashayam* - *Sarabendhira vaithiya rathnavali*, pg no: 74

Birungamala thailam - *Sarabendhira vaithiya rathnavali*,pg no: 184

Thaalisa chooranam - *Theraiyar maga karisal,* pg no: 114

*Maga vilvaadhi legiyam* - *Sarabendhira vaithiya rathnavali*,Part 2 pg no: 184

***Panai vithu*** - *Neerizhivuku marundhu*

*Sarabendhira vaithiya rathnavali,* pg no: 383

***Panai matai*** is one of the ingredients of following medicines:

*Nayana rogathirku Maga nirkundi thailam - sarabendhira vaithiya muraigal,*

Pg no: 31

*Birungamalaga thailam* - *sarabendhira vaithiya muraigal,* Pg no: 19

*Elaadhi nei* - *sarabendhira vaithiya muraigal,* Pg no: 41

*Nayana rogathirku mathirai* - *sarabendhira vaithiya muraigal*, Pg no: 64

*Poo Padalathirku mathirai* - *sarabendhira vaithiya muraigal,* Pg no: 65

*Nayana viyadhiku urundai* - *sarabendhira vaithiya muraigal*, Pg no: 80

*Piramiyam theera* - *Agathiyar atavanai vaagadam,* Pg no: 52

*Sevvatai kadiku chooranam* - *Agathiyar atavanai vaagadam*, Pg no: 87

## 3.3 SIDDHA ASPECT OF THE DISEASE

**Peptic ulcer - *Gunmam***

**The essential features of the disease are**

The food eaten will not be digested, burning sensation and pain will be produced. Vomiting also occurs so that the food eaten becomes useless. As a result, the nutritional status and strength of body diminish day by day. Patient may also develop depression due to the same and may develop suicidal tendency.

In one type of this disease, there will be flatulence. The air will move like a ball inside the abdomen and may produce great discomfort. Hence some authors call it as *kulmam.*

நோய் வரும் வழி :

கயமான குடலி னுள்ளே
கல்லுமி நெல்லு மாமே
கல்லொரு மயிரா யுள்ள
கசடது குடலிற் பற்றி
வல்லபாங் கதுவா யன்னஞ்
செரியாத மாசி னாலே
மெல்லிய கிருமி கொண்டு
குன்மநோய் மருவுங் காணே.

பரராச சேகரம்

செய்யான குன்மத்தின் தோற்றந் தன்னைச்
செப்பிடவே துவர்ப்பான பொசிப்பி னாலும்
மெய்யான மங்கையுடன் மருவ லாலும்
வகையாகுங் கிழங்குவகை யருந்த லாலும்
உய்யான மிளகுவகை உரைப்பி னாலும்
உறுபசியை யடக்கிடும் மந்தத் தாலும்
தய்யாள சண்டாள கோபத் தாலும்
சலிப்பாலும் குன்மம்வந் தடையும் பாரே.

யூகி சிந்தாமணி

**Genesis of the disease**

It is considered that the disease may develop due to the following factors:

- By eating excessively hot substances and substances which produce belching.
- By eating dietary items which are mixed with sand, bran, stone, and dust.
- By drinking spring water, stagnant water, lime mixed water.
- By eating excess of diets which will not be easily digested such as coconut milk.

- By getting angry, starvation, and depression frequently.
- In addition, it is controllable that those who practice yoga by improper controlling of breath, may also develop the disease.

**Prodromal symptoms**

Anorexia and even if there is appetite,dislike to food are the main features.

In addition, there may be nausea, frequent flatulence, excessive salivary secretion, regurgitation of ingested food, abdominal pain due to increased peristalsis, vomiting, flatulence with increased bowel sounds.

**Types of the disease**

As per the opinion of the saint *Yugi* the disease has been classified into eight types as follows:

1. *Vayu gunmam*
2. *Vatha gunmam*
3. *Pitha gunmam*
4. *Eri gunmam*
5. *Vali gunmam*
6. *Sathi gunmam*
7. *Sanni gunmam*
8. *Sethuma gunmam*

However, another according to the saint *Thirukanda munivar* classified it is into eight different types as follows:

1. *Vatha gunmam*
2. *Pitha gunmam*
3. *Kapha gunmam*

4. *Vatha pitha gunmam*
5. *Vatha kapha gunmam*
6. *Pitha silethuma gunmam*
7. *Tridosha gunmam*
8. *Raththa gunmam*

Further, he has subdivided *ratha gunmam* into two subtypes as

1. *Ratha vatha gunmam*
2. *Ratha vatha pitha gunmam*.

Some other ancient physicians have classified the disease into 3 types as follows:

1. *Saamaniya vatha gunmam*
2. *Saamaniya pitha gunmam*
3. *Saamaniya silethuma gunmam*

In some ancient textbooks in addition to four types of *gunmams* associated with *doshas*,

1. *Paayuru gunmam*
2. *Eri gunmam*
3. *Vaandhi gunmam*
4. *Vali gunmam* have also been included, thus totaling eight.

Of these, it appears that paayuru gunmam is the gunmam mentioned as *vayu gunmam* by the saint yugi. The term *paayuru* will denote anus and the disease is caused by the excessive activity of downward directional factor (Abana *Vayu*). It appears that because of this, the disease was also called as *vayu gunmam*; the signs and symptoms of the disease are similar to *soolai gunmam*, hence it may be stated that both are one and the same.

In northern text books, three *gunmams* associated with *dosha* (*vatha, pitha and kapha*) have been mentioned. In addition, *vatha pitha*, *Vatha Pitha* and *pitha kapha* which originate from the combination of two have also been mentioned.

Besides, *Sanni gunmam* and the *ratha gunmam* developed due to inflammation of the ovaries in women have also been included, thus totaling eight.

Saint *Thirumoolar* has mentioned that three numbers of *gunmam* (*Vatha gunmam, Soolai gunmam* and *Vali gunmam*) were developed due to *vatha dosha*.

It has also been mentioned that *eri gunmam*, *vaanthi gunmam* and *azhal* (*Pitha) gunmam* manifest due to *pitha dosha*; he has also mentioned that *kapha gunmam* and *tri dosha gunmam* (*Muppini gunmam* ) manifest due to *Kapha dosha*; it is hoped that *Thirumoolar*'s suggestion may help to simplify the treatment.

The *ratha gunmam* mentioned above is specific to women as felt by the saint *yugi*. It is felt that it would be proper if it detailed in the section on women's special diseases.

Even though *Thirumoolar* considered the eight types of *gunmam* into four types only from *dosha* point of view, they were not detailed here in order to simplify things.

***Vali gunmam***

In this disease, the food ingested will not be digested properly and produces stomach pain as if there is some pathology in the stomach.

Even though the patient will not take food properly, the body will appear as obese. However, patient will loose his strength and will become lean; he may not even in a position to walk.

There will be also pain in head and in whole of the bode; patient may also develop tiredness, giddiness, thirst and dryness of tongue.

In addition, the body will become black in colour with dryness.

The disease mostly appears during the age of 20 to 30 years which is the *vatha* period.

Later, may patient may develop severe abdominal pain. Vomiting may also follow which will reduce the pain slightly. There may be also small quantity of blood in the vomit which appears dark in colour.

As the days pass, the disease will progress and produces indigestion throughout the life of the patient.

Even though the patient likes to take food, he will develop unbearable pain in the abdomen and over the corner of the chest below the xiphisternal region within few hours due to the fact that the food ingested will not get digested.

Sometimes there may be burning pain in the stomach during night. Even though the patient will feel like having appetite either he may not take food due to the fear of pain or he may eat and try to induce vomiting with finger, these clinical features also will continue.

***Azhal Gunmam***

Due to the formation of excessive *pitha dosha* in this disease, the patient will be having burning pain in the stomach.

He will also develop nausea and vomiting. The vomitus may be found mixed with nucus and *pitha*. Patient may become unconscious after vomiting. As the vomiting increase, the burning pain in the chest and abdomen also increase.

In addition, there will be pain in the chest and abdomen also increase. In addition, there will be pain in the upper abdomen and the urine will appear red in colour.

Excessive thirst, vertigo are other features of the disease.

Besides, there will be loss of body strength and impaired quality of blood as the foods taken or vomited out; this causes yellow colouration of the skin.

Sometimes, the *pitha dosha* causes uncontrollable pain in the inflamed stomach and produces uncontrollable vomiting.

The upper abdomen will be distended with gas; the food ingested will be digested or indigested and come out along with blood; further the body will appear warm and there may be headache; burning sensation of eye and fatigue may also occur.

As the disease progresses the pain in the abdomen will be severe after eating. The abdomen will appear heavy, the appetite will be impaired and patient will dislike to eat.

Ageusia, dryness of tongue, burning pain over the upper stomach, regurgitation of fluid in the stomach, tiredness, laziness, head disease, insomnia and sour belch from the stomach are other features of the disease.

In addition, the body will become lean and patient may develop dyspnoea. It is considered that the disease occurs in the age group of 30 to 50 years.

***Iya gunmam***

The essential features of the disease are; dislike to food, atrophy of mouth, lean body mass due to malnutrition leading to anaemia, vertigo, irritation of the throat, tremors and frequent fright.

If the disease occurs in the old age period which is the period of 'kapha Period', lot of adverse effects may develop.

The ingested food will not get digested and stays in the stomach; the food will get fermented and come out only in the vomitus with a smell of meat.

The food will never pass down the anus, the abdomen will be distended like a drum and there will be belching, the abdomen will be distended with fluids and makes sound when the body moves, patient may also develop vomiting of white coloured substances frequently.

The gas accumulated in the stomach may also roll like a ball.

Besides the above features, there may be also continuous pain in the abdomen and excessive haemetemesis. The body also may become lean day by day; these feautures will make one to suspect that patient might be having cancer in stomach.

***Tri –Dosha (Sanni) gunmam***

In this type of disease, patient may not have the desire for food.

There will be excessive salivary secretion, the abdomen will be distended and motion will be passed hot with excessive sound, the mouth will have salty taste, there will be also irritation of the throat, the patient will frequently develop belch, dyspnoea and giddiness, the body also will become cool.

***Kaal (Vayu) Gunmam***

The disease may be also called as *paayuru gunmam* and *soolai gunmam*.

In thise disease, the food ingested will not be digested and will come out in the vomit.

The patient may also develop dislike to food. Even though the food ingested will be of small quantity, abdomen will appear distended like gas filled balloon, there will be loss of body strength, and the patient may also have rest due to inability to walk.

There may be also sweating of the body. Patient may also develop unbearable pain in the stomach due to gas in the same producing bow like pulling sensation.

The excessive activity of *Vatha dosha* in this disease causes damage to the stomach. The downward directing factor (*Abana vayu*) gets stimulated and it prevents the digestion of ingested food, this results in abdominal pain, irritation of chest, pain as if the intestine is twisted.

***Eri Gunmam***

In this type of disease, unbearable irritation in the stomach develops within a short time after taking the food.

The patient will develop pain as if the stomach is twisted.

Excessive salivary secretion, pain in the head, sour belch from the stomach, distention of abdomen with excessive bowl sound, diarrhoea and sweating over the root of hair or other features of the disease.

Ultimately the patient will become lean.

***Vaanthi* (Vomiting *Gunmam*):**

In this disease, the patient will develop indigestion, vomiting, giddiness, burning pain in stomach, spasm and irritation of stomach, constipation, a sensation of heat of fire in the body, inability to walk and ageusia.

In addition there will be protuberance of nerves and patient may develop numbness. Ultimately the patient will develop loss of strength.

***Vali Gunmam***

In this disease, the food ingested will not be digested and the abdomen will be distended with gas.

The body skin will shrink and atrophy.

Depression, insomnia, dislike to food, diarrhea with excessive bowl sounds, pain at the rib sides as if it is pricked by thorn, pain in the hip and in the vertebral region, throbbing pain of the whole body, frequent attacks of fever and pseudo-appetite are the other features of the disease.

Besides the above features, as the *kapha dosha* and *vatha dosha's* are associated in this disease, the ingested food will not be digested and also will not be vomited out.

The food stays in the stomach and causes pricking pain in throat and twisting pain in stomach.

Sometimes, the patient may not be in a position to bear the pain and even develop suicidal tendency, there where patients who had even commited suicide.

**General features of the disease**

The disease usually occurs in men in the age group of 25-45 years.

However, the disease may also occur in women and also in the older age.

When patients have good body strength and capacity to digest any type of food, may suddenly develop anorexia, nausea, bilious vomiting, belch and soar belch from the stomach.

The disease progresses gradually and other features such as indigestion, appearance as if the abdomen is distended, rolling pain in the abdomen may also appear.

As the disease increases in severity, patient may develop unbearable pain and may put the finger into the throat to induce vomiting, after vomiting the pain may be reduced slightly; some patients may roll over the cot and cry due to excessive pain and even will commit suicide.

***Doshas* and other factors**

As per the saying of ancient saint 'Theran' *gunmam* may not develop if the (*Vadha bandha malathu gunmam varadhu*) due to change in food habits, the *vadha dosha* worsens.

The other *doshas* will also be associated with it and fail to perform their natural functions. As a result of this, the downward directional factor (*Abana vayu*) and the upward directional factor *(Udhana Vayu*) were malfunctioning.

The food ingested will not be digested and purity of blood is also lost.

In addition to the above, the downward directional factor controls the passage of stool and causes increase of gas in the stomach;The upward directional factor causes vomiting and aggravate the disease.

**Pulse**

If the *vatha* pulse runs in the left side or by the side, it may denote *vatha gunmam*.

Similarly it has been stated that if the other types of pulses do not move in their proper direction and move to the left or by the side, they may give glue to appropriate *gunmam* disease.

If the *vatha* pulse and *Pitha* pulse appear to be associated and their strength also appears to be integrated as single strength, it can be considered that this denotes *Eri gunmam* due to *mantham*.

If the *vatha* pulse is felt abnormally like a vibration of tightly held rope which has raised with a finger and then suddenly dropped, it may be suggestive of *Vali gunma* disease.

## 3.4 MODERN ASPECT OF THE DISEASE

**Peptic ulcer disease**

Peptic ulcer disease comprises ulcers in the stomach or duodenum.

Ulceration of the gastro intestinal tract due to the combined action of hydrochloric acid and pepsin. This occurs most commonly in either the stomach or duodenum but it may also occur in the oesophagus, Meckel's diverticulum, jejunum.

**Aetiology**

1. Imbalance between the damaging effects of acid and pepsin attack and the body's mucosal diffences.

2. Four factors may account for the tendency to hyper secrete acid and pepsin.

    a. Increased parietal cell mass

    b. Increased stimulation of acid secretion.

    c. Increased parietal cell sensitivity to stimulants

    d. Decreased inhibitory control of acid secretion.

3. Psychological factors – Stress of modern life is often invoked as an important cause.

4. Epidemiological factors:

    a. Ulcer is more common in men than women. Ulcer is more frequent in women after the menopause, suggesting that sex hormones may be important in the aetiology of the disease.

    b. Duodenal ulcers more common in patients with blood group "O". Gastric ulcers more common in patients with blood group 'A'

5. A major causative factor (60% of gastric and up to 90% of duodenal ulcers) is chronic inflammation due to ***Helicobacter pylori*** that colonizes the antral mucosa.

6. Other important contributing factors are NSAIDs.

7. Additional aggressive factors include smoking, ethanol, bile acids, aspirin, and steroids.

8. Hyper parathyroidism is also associated with increased incidence of peptic ulcer and it is now known that a raised serum calcium level in man stimulates gastric secretion.

**Pathophysiology**

PUD develops when the mucosa of the GI Tract becomes susceptible to corrosive forces, the most important of which is gastric acid.

The DU patient is an acid hypersecretor. Meal stimulated gastric acid secretion was approximately 70% higher, while night (basal) secretion was approximately 150% higher. The presence of food appears to protect the gastric mucosa against the meal- induced hyperchlorhydria.

At night, however the increased quantity of acid is left to bathe the 'bare' gastroduodenal mucosa.

Duodenal bicarbonate secretion appears to be impaired in DU patients.

When the balance between protective factors (Prostaglandins, mucus secretion, and nitric oxide) and aggressive factors (gastric acid, pepsin, and bile salts) is the disrupted, mucosal injury may ensue.

The three main factors that contribute to disturbing this balance are H.pylori infection, NSAID use, and smoking.

**HELICOBACTER PYLORI (H. pylori)**

H. pylori is a gram negative, spiral, flagellated, urease positive bacterium that has the ability to colonize the gastroduodenal mucosa and cause inflammation.

By virtue of its ability to split urea into bicarbonate and ammonia, the organism is able to buffer $H_+$ ions before the intracellular pH drops below 5, allowing it to survive in the acidic environment of the stomach and adhere to specific receptors on the gastric epithelial cell.

Once attached , the organism causes mucosal damage by various mechanisms.

**Non steroidal anti inflammatory drugs**

NSAIDs produce topical mucosal injury due to their acidic nature, with additional damage due to the biliary secretion and subsequent duodenogastric reflux of active NSAID metabolites.

The principal toxicity of NSAIDs occurs as a result of their systemic ability to impair prostaglandin synthesis.

**Cigarettes**

Smoking adversely affects many of the natural mucosal protective mechanisms and enhances aggressive factors, such as stimulating pepsin release and increasing the production of free radicals.

Smoking also appears to increase the risk and virulence of H.pylori.

**Hypersecretory states**

In some patients, the balance between protective and aggressive factors is disrupted because of the overproduction of acid due to hypersecretory state.

The most noteworthy of these conditions is Zollinger- Ellison syndrome, in which a gastrin secreting tumor stimulates acid production that is not subject to the normal feedback inhibition of acid.

Other rare causes of hypersecretory states are systemic mastocytosis, antral G-cell hyperplasia, and extensive small bowel resection.

**Symptomatology**

1. The main symptom is pain which is usually localized to the epigastrium.
2. Duodenal ulcers pain occurs 1-3 hours after a meal and may awaken patient from sleep. Pain is relieved by food, antacids.
3. In Gastric ulcers food may exacerbate the pain while vomiting relieves it.
4. Waterbrash which seems to be caused by an increased salivary secretion.
5. Bloating
6. Dyspepsia
7. Belching
8. Nausea

9. Vomiting

10. Hematemesis and melena may also occur in chronic condition.

[ Ref: Oxford Text book of Medicine, Ronald's text book of Medicine, and The Clinician's guide to Acid/peptic Disorders of Gastrointestinal Tract ]

## 3.5 LATERAL RESEARCH OF *Borassus flabellifer*

1. Anti-inflammatory activity of ethanolic extract of *Borassus flabellifer* l. Male flowers Mahesh S. 2009

2 . Evaluation of the analgesic and antipyretic activities of ethanolic extract of male flowers (inflorecences) of *Borassus flabellifer* l. Mahesh s. 2009

3. Anti nociceptive, anti inflammatory and anti bacterial properties of leaf of female *Borassus flabellifer* (Arecaceae) Sandhya *S et al* 2010

4. Preliminary studies on in vitro anti-tumor activity of tender seed of *Borassus flabellifer* Govinda Rao Duddukuri et al

5. Antibacterial activity of methanolic seed coat extract of *Borassus flabellifer* l. Govinda Rao Duddukuri et al.

6. Bio active saponins from the flowers of *Borassus flabellifer* Yoshikawa masayuki et al.

The methanolic extract from the flowers of *B. flabellifer* was found to show inhibitory effect on serum glucose elevation. By bioassay-guided separation, six new spirostanol saponins 1, 2, 3, 4, 5, and 6 were isolated from the active fraction (BuOH-soluble fraction) together with 21 known furostanol and spirostanol saponins. In addition, the principal constituent, dioscin (7), exhibited inhibitory effect on serum glucose elevation in oral sucrose-loaded rats as well as protective effect on gastric mucosal lesions induced by ethanol in rats.

# 4. MATERIALS AND METHODS

## 4.1. PREPARATION OF *CHOORANAM*

### Collection and authentication of the materials

The plant material used in this study was collected during the month of January - February (2012) from Soorapalli, Salem district, Tamilnadu, India and authenticated by Botanist , Central Research Institute for Siddha and Siddha experts of Gunapadam Dept . The drug "***Panaipoo"*** was selected from the classical Siddha literature *Agathiyar gunavaakadam, Gunapadam mooligai vagupu.*

### Preparation of *Panai poo Chooranam:*

***Panai poo*** was thoroughly cleaned to remove impurities. Then it was cut into small pieces and dried. Later they were finely powdered. The resultant powder was subjected to sieve with white cotton cloth to obtain finest physical form. (*Vasthirakayam)*

### Purification of *chooranam*

The *Chooranam* was moistened with cow's milk. Pot was filled with milk and water of equal ratio to nearly ¾ its volume. The mouth of the pot was covered and tied with white cotton cloth. The *Chooranam* moistened by milk was placed above the tied cloth. The mouth of the pot was closed with another mud pot. The gap between the two mud pots was tied with a wet cloth to avoid evaporation. Then this pot was subjected to heat and boiled until milk level in lower pot gets reduced to ¼ volumes. Then the powder was taken, sun dried, powdered finely and preserved for usage.

### Preservation

The purified *Chooranam* was stored in a clean, air tight glass container.

**Figure No:4.1 *Panai poo chooranam***

**Life span**

3 Months.

**Administration of the drug**

| | |
|---|---|
| Form of the medicine | : Chooranam |
| Route of Administration | : Enteral |
| Dose | : 1 gm |
| Anubanam (Vehicle) | : Warm water |
| Times of Administration | : Two times a day; before food |

## 4.2. STANDARDIZATION OF *PANAI POO* CHOORANAM

Standardization of drugs means confirmation of its identity and determination of its quality, effectiveness and acceptability to be used as medicine. Standardization of plant drug is based on the concentration of their active principles, physical and chemical standards. Plant drug has been standardized on the basis of organoleptic properties, physical characteristics, and physico-chemical properties. The process of standardization can be achieved by stepwise studies.

**Collection and identification of plant**

The plant material belongs to the family Arecaceae was collected from Soorapalli, Salem district, Tamilnadu. The plant was identified with the help of Botanist, Central Research Institute for siddha, Chennai and by the Siddha experts of Gunapadam Dept. A voucher specimen is deposited in the Herbarium, Department of Gunapadam , Govt.Siddha Medical Collage, Chennai-106.

### 4.2.1. PHARMACOGNOSTIC STUDY

***Borassus flabellifer* Linn**

**Materials and methods for anatomical studies**

**Collection of specimens**

The plant specimens for the proposed study were collected from Soorapalli, Salem Dist, Tamilnadu. Care was taken to select healthy plants and normal organs. The required samples of different organs were cut and removed from the plant and fixed in FAA (Farmalin-5ml+ Acetic acid-5ml + 70% Ethyl alcohol-90ml).After 24 hrs of fixing, the specimens were dehydrated with graded series of tertiary –Butyl alcohol as per the schedule given by Sass, 1940. Infiltration of the specimens was carried by gradual addition of paraffin wax (melting point 58-60 C) until TBA solution attained super saturation. The specimens were cast into paraffin blocks.

**Sectioning**

The paraffin embedded specimens were sectioned with the help of Rotary Microtome**.** The thickness of the sections was 10-12 μm. Dewaxing of the sections was by customary procedure (Johansen, 1940). The sections were stained with Toluidine blue as per the method published by O'Brien et al. (1964). Since Toluidine blue is a polychromatic stain. The staining results were remarkably good; and some cytochemical reactions were also obtained. The dye rendered pink colour to the cellulose walls, blue to the lignified cells, dark green to suberin, violet to the mucilage, blue to the protein bodies etc. wherever necessary sections were also stained with safranin and Fast-green and IKI(for Starch)

For studying the stomatal morphology, venation pattern and trichome distribution, paradermal sections (sections taken parallel to the surface of leaf) as well as clearing of leaf with 5% sodium hydroxide or epidermal peeling by partial maceration employing Jeffrey's maceration fluid (Sass, 1940) were prepared. Glycerine mounted temporary preparations were made for macerated/cleared materials. Powdered materials of different parts were cleared with Naoh and mounted in glycerine medium after staining. Different cell component were studied and measured.]

**Photomicrographs**

Microscopic descriptions of tissues are supplemented with micrographs wherever necessary. Photographs of different magnifications were taken with Nikon labphoto 2 microscopic Unit. For normal observations bright field was used. For the study of crystals, starch grains and lignified cells, polarized light was employed. Since these structures have birefringent property, under polarized light they appear bright against dark background. Magnifications of the figures are indicated by the scale-bars. Descriptive terms of the anatomical features are as given in the standard Anatomy books (Esau, 1964).

**4.2.2 Physico-chemical Investagations** :

Physico-chemical studies like total ash, water soluble ash, acid insoluble ash,

water and alcohol soluble extract, loss on drying at 105°C and $_{P}H$ as per the WHO guide lines in Central Research Institute For Siddha.

**Determination of Total Ash**

Incinerate about 2 to 3 g accurately weighed, of the ground drug in a tared platinum or silica dish at a temperature not exceeding 450º until free from carbon, cool and weigh. Calculate the percentage of ash with reference to the air-dried drug.

**Determination of Acid Insoluble Ash**

Boil the ash obtained for 5 minutes with 25 ml of dilute hydrochloric acid; collect the insoluble matter in a Gooch crucible or on an ash less filter paper, wash with hot water and ignite to constant weight. Calculate the percentage of acid-insoluble ash with reference to the air dried drug.

**Determination of Alcohol Soluble Extractive**

Macerate 5 g of the air dried drug, coarsely powdered, with 100 ml of Alcohol of the specified strength in a closed flask for twenty-four hours, shaking frequently during six hours and allowing to stand for eighteen hours. Filter rapidly, taking precautions against loss of solvent, evaporate 25 ml of the filtrate to dryness in a tarred flat bottomed shallow dish, and dry at 105º, to constant weight and weigh. Calculate the percentage of alcohol-soluble extractive with reference to the air-dried drug.

**Determination of Water Soluble Extractive**

Proceed as directed for the determination of Alcohol-soluble extractive, using chloroform water instead of ethanol.

**Determination of Moisture Content (Loss on Drying)**

Procedure set forth here determines the amount of volatile matter (i.e., water drying off from the drug). For substances appearing to contain water as the only volatile constituent, the procedure given below, is appropriately used. Place about 10 g of drug (without preliminary drying) after accurately weighing (accurately weighed to within 0.01 g) it in a tared evaporating dish. For example, for underground or unpowderdd drug, prepare about 10 g of the sample by cutting shredding so that the parts are about 3 mm in thickness.

After placing the above said amount of the drug in the tared evaporating dish dry at 105º for 5 hours, and weigh. Continue the drying and weighing at one hour interval until difference between two successive weighings corresponds to not more

than 0.25 per cent. Constant weight is reached when two consecutive weighings after drying for 30 minutes and cooling for 30 minutes in a desiccator, show not more than 0.01 g difference.

**Determination of pH**

1% solution of plant drug was prepared in distilled water and pH was determined using pH meter, systronics digital ph meter, MK VI.

**TLC estimation of *Panai poo* Chooranam:**

Thin-layer chromatography is a technique in which a solute undergoes distribution between two phases, stationary phase acting through adsorption and a mobile phase in the form of a liquid. The adsorbent is a relatively thin, uniform layer of dry finely powdered material applied to a glass, plastic or metal sheet or plate. Glass plates are most commonly used.

Identification can be effected by observation of spots of identical Rf value and about equal magnitude obtained, respectively, with an unknown and a reference sample chromatographed on the same plate. A visual comparison of the size and intensity of the spots usually serves for semi-quantitative estimation.

Solvent system:

Toluene : Ethyl acetate (6:1.5).

TLC plate:

Aluminium plate precoated with silica gel $60F_{254}$ of 0.2 mm thickness (Merck).

Developing chamber:

Camag's twin trough chamber.

Visualizing reagent:

Vanillin-sulphuric acid agent.

Extract Preparation:

4 g of the chooranam was soaked overnight in chloroform. Boiled on a water bath for 10 mins, filtered and concentrated to 10 ml.

Procedure:

The extract was applied on the TLC using capillary and developed in the solvent system. The developed TLC plate was air dried, dipped in vanillin-sulphuric acid reagent and heated in an oven at 105°C until the development of coloured spots and photograph taken.

**4.2.2.3 Fourier transform infrared spectroscopy (FTIR):**

**Instrument details:**

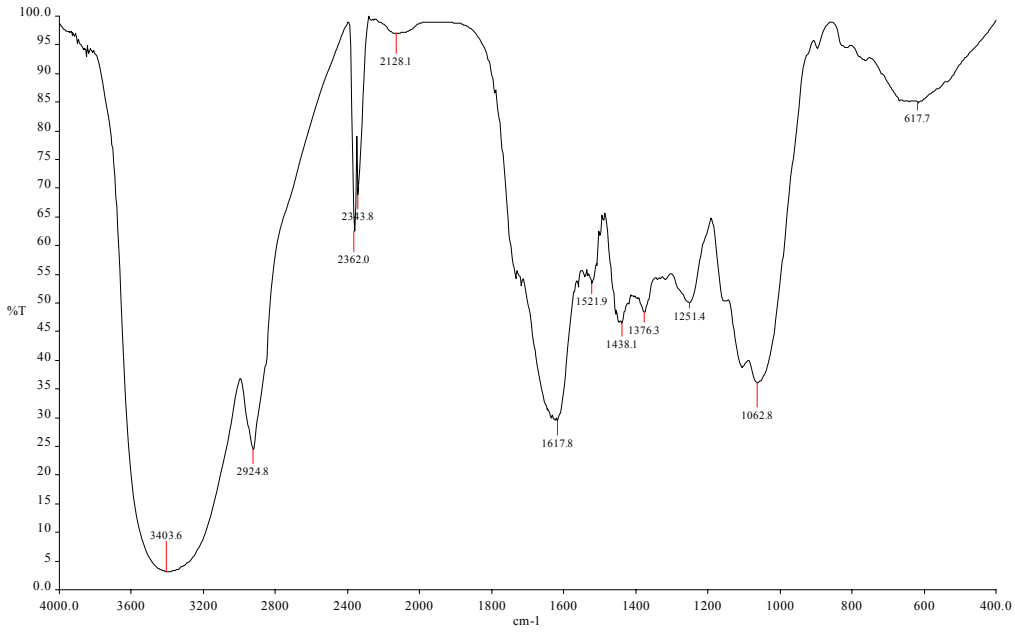
| | |
|---|---|
| Model | : Spectrum one: FT-IR Spectrometer |
| Scan Range | : MIR 450-4000 cm-1 |
| Resolution | : 1.0 cm-1 |
| Sample required | : 50 mg, solid or liquid. |

Fourier Transform Infrared Spectroscopy (FTIR) is an analytical technique used to identify mainly organic materials. FTIR analysis results in absorption spectra which provide information about the chemical bonds and molecular structure of a material. The FTIR spectrum is equivalent to the "fingerprint" of the material and can be compared with cataloged FTIR spectra to identify the material.

Infrared spectrum is useful in identifying the functional groups like -OH, -CN, -CO, -CH, -NH2, etc. Also quantitative estimation is possible in certain cases for chemicals, pharmaceuticals, petroleum products, etc. Resins from industries, water and rubber samples can be analyzed.

The drug sample was analyzed by the FTIR to identify the chemical bonds and molecular structure of a material.

**Figure No: 4.2.2.3**




### 4.2.2.4 Scanning electron microscope (SEM):

The Scanning Electron Microscope (SEM) is a microscope that was electrons rather than light to form an image. There are many advantages to using the SEM instead of a light microscope.

Resolution : 1.2 nm gold particle separation on a carbon substrate

Magnification : From a min of 12 x to greater than 1, 00,000 X

The SEM has a large depth of field, which allows a large amount of the sample to be in focus at one time. The SEM also produces images of high resolution, which means that closely spaced features can be examined at a high magnification. Preparation of the samples is relatively easy since most SEM one require the sample to be conductive.

The combination of higher magnification, larger depth of focus, greater resolution, and easy of sample observation marks the SEM one of the most heavily used instruments in research areas today.

### 4.2.3. Qualitative phytochemical analysis:

**Table No:4.2.3**

| Sl. No | EXPERIMENT | OBSERVATION | INFERENCE |
|---|---|---|---|
| 1. | **Test for Alkaloids:** Alkaloids are identified by precipitate method **Dragendroff's reagent:** An aqueous plant extract of 2 ml is mixed with 2 ml of Dragendroff's reagent. | Absence of reddish brown precipitate | Absence of alkaloids |
| 2. | **Test for Triterpenoids (Noller's Test)** To few mg of extract, add tin and thionyl chloride and heat in water bath. | Presence of purple colour | Presence of Triterpenes |
| 3. | **Test for Tannins:**A plant sample dried powder 0.5 gm is boiled in 20 ml of water and filtered. The filterate 2 ml is taken and 3-5 drops of $Fecl_2$ (0.1%) is slowly added to it. | Forms a brownish-green or bluish-black colour. | Presence of Tannins |
| 4. | **Test for Flavonoids:** An aqueous filterate of plant sample is mixed with 5 ml of dilute Ammonium solution and few drops of concentrated $H_2SO_4$ is slowly added through the sides of the test tube. | Absence of Yellow colour formed | Absence of flavonoids |
| 5. | **Test for steroids:**An ethonolic extract of plant sample 2ml is mixed with 2 ml $H_2SO_4$ and 0.5 gm Acetic anhydride. | The solution turns in to blue to green colour | Presence of Steroids |

| Sl. No | EXPERIMENT | OBSERVATION | INFERENCE |
|---|---|---|---|
| 6. | **Test for Saponin:** A powdered 2 gm of plant sample is boiled with 20 ml of distilled water, then filtered, the filterate is added with fresh 5 ml of distilled water and shaken vigorously. | A permenant or persistant froth is formed. The froth is turned in to emulsion by adding three drops of olive oil. | Presence of saponin |
| 7. | **Test for Phenolic compounds:** About 2 ml of aqueous plant extract is mixed with 2 ml of $Fecl_3$ solution. | Absence of deep bluish green colour | Absence of phenolic compounds |
| 8. | **Test for Glycosides:**An aqueous plant extract of 2 ml is added with 1 ml of concentrated Hcl and boiled in a water bath for about 2 hours. The filtrate is separated and 3 ml of chloroform is added, then 10% of 1ml Ammonium solution is added. | Pink colour formed. | Presence of glycosides |

### 4.2.4. Chemical analysis

**Proximate Chemical Analysis of *Panai poo chooranam***

**Methodology For Chemcial Analysis**

**Preparation of Extract**

Add 5 gm of the sample to 50ml of distilled water. Boil the solution for 20 minutes, cool and then filter. The Extract was used for the following tests.

**Table No: 4.2.4**

| S.No | Experiment | Observation | Inference |
|---|---|---|---|
| 1. | **Test for reducing Sugar :** To 5ml of Benedicts qualitative reagent, add 10 drops of extract, then boil for two minutes | Green colour PPT | Presence of Reducing Sugar |
| 2. | **Test for Starch :** To 5 ml of extract add 2ml of acetic acid and then add few drops of N/50 Iodine Solution. | Blue Colour | Presence of Starch |
| 3. | **Test for Proteins :** To 2 ml of extract, add 2ml of 5% Sodium Hydroxide mix and add 2 drops of Copper Sulphate Solution. | Violet Colour | Presence of Proteins |
| 4. | **Test for amino Acid :** Place 2 drops of extract on a filter paper and allow to dry well. Then spray 1% ninhydrin over the same and allow to dry. | Violet Colour | Presence of Amino Acid |
| 5. | **Test for Albumin :** To 2 ml of extract, add 2ml of Esboch's reagent. | Yellow PPT | Presence of Albumin |
| 6. | **Test for Phosphate :** To 2ml of extract, add 2ml of ammonium Molybdate solution and 2ml of concentrated Nitric Acid. | Absence of yellow PPT | Absence of Phosphate |
| 7. | **Test for Sulphate :** To 2 ml of extract add 2ml of 4% ammonium oxalate solution. | Absence of white PPT | Absence of Sulphate |
| 8. | **Test for Chloride :** Add 2ml of extract to dilute nitric acid till the effervescence ceases. Then add 2 ml of Silver Nitrate Solution. | Cloudy White PPT | Presence of Chloride |
| 9. | **Test for Iron :** To 2ml of extract, add 2ml of ammonium thio cynate solution and add 2ml of concentrated Nitric Acid. | Red Colour | Presence of Iron |
| 10. | **Test for Calcium :** To 2 ml of extract, add 2 ml of 4% ammonium Oxalate Solution. | Absence of white PPT | Absence of Calcium |

| S.No | Experiment | Observation | Inference |
|---|---|---|---|
| 11. | **Test for Sodium :**<br>Make a paste with 2 pinches of the sample with Hcl and Introduce it into the blue flame. | Absence of yellow Flame | Absence of Sodium |
| 12. | **Test for Potassium :**<br>Add a pinch of the sample to 2 ml of Sodium Nitrate Solution. Then add 2ml of Cobal Nitrate in 20% acetic acid. | Absence of yellow PPT | Absence of Potassium |
| 13. | **Test for Zinc :**<br>To 2ml of extract, add few drops of Sodium Hydroxide. | Absence of white PPT | Absence of Zinc |
| 14. | **Test for Magnesium :**<br>To 2ml of extract, add few drops of Sodium Hydroxide Solution | Absence of white PPT | Absence of Magnesium |
| 15. | **Test for Alkaloids :**<br>a. To 2ml of extract, add 2ml of Potassium Iodide Solution<br>b. To 2ml of extract add 2ml of Picric Acid.<br>c. To 2 ml of extract add 2ml of Phosphotunqstic Acid. | Absence of red Colour<br>Absence of yellow Colour<br>Absence of white PPT | Absence of Alkaloids<br>Absense of Alkaloids<br>Absence of Alkaloids |
| 16. | **Test for Tannic Acid :**<br>To 2ml of extract add 2 ml of Ferric Chloride Solution | Black PPT | Presence of Tannic Acid |

## 4.3. TOXICOLOGICAL STUDY

### MATERIALS AND METHODS

**Chemicals and reference drug:** All chemicals used in the present study were analytical grade and purchased from SD fine chemicals Ltd (Mumbai, India). Aspirin was obtained from BD Pharmaceutical Works and Ranitidine (Reference drug) was obtained from Ranbaxy Laboratories.

***Stock solution preparation***

The powdered form of Panai Poo Chooranam was mixed uniformly in 2% CMC solution to achieve 100mg/ml as main stock solution and used in this study.

***Animals***

Albino mice of either sex weighing 25-30g (For acute toxicity study) and Healthy Swiss Albino rats of the Wister strain weighing 150-200 g were used for the study. The animals were used with the approval of the Institute animal ethics committee and obtained from Vels University, Chennai. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-28$^0$C temperature, standard light cycle (12 h light, 12 h dark) and water ad libitum.

Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation. The rats were randomly assigned to control and different treatment groups, six animals per group.

(Approval number: XIII/VELS/PCOL/11/2000/CPCSEA/IAEC/08.08.2012). The animals were acclimatized for one week under laboratory conditions.

**Acute toxicity study**

Acute oral toxicity test for the ***Panai poo Chooranam*** was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice. Single animals are dosed in sequence usually at 48 h intervals.

However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal. The time interval was adjusted as appropriately in case of inconclusive response. The test is simpler to implement when a single time interval is used for making sequential dosing decisions. Special attention was given during the first 4 hours and daily thereafter, for a total of 14 days, except where they need to be removed from the study and humanely killed for animal welfare reasons or are found dead.

All observations are systematically recorded and Observations include changes in skin and fur, eyes and mucous membranes, and also respiratory, circulatory, autonomic and central nervous systems, and somatomotor activity and behaviour pattern. Attention was directed to observations of tremors, convulsions, salivation, diarrhea, lethargy, sleep and coma. The principles and criteria summarized in the Humane Endpoints Guidance Document taken into consideration. Animals found in a moribund condition and animals showing severe pain or enduring signs of severe distress was humanely killed. When animals are killed for humane reasons or found dead, the time of death was recorded.

## 4.4. PHARMACOLOGICAL STUDY

### EVALUATION OF ANTIULCER PROPERTY OF *PANAI POO CHOORANAM*

Gastric and duodenal ulcers are illnesses that affect a considerable number of people in the world. Some of the causes of these disorders are: stress, smoking, nutritional deficiencies and ingestion of non-steroidal anti-inflammatory drugs. The pathogenesis of gastro duodenal ulcers are influenced by various aggressive and defensive factors, such as acid-pepsin secretion, mucosal barrier, mucus secretion, blood flow, cellular regeneration and endogenous protective agents (prostaglandins and epidermal growth factor).

The genesis of peptic ulcer mechanism is related to the balance between the defensive and aggressive factors. Gastric mucosa is an active area of arachidonic acid metabolism the integrity of gastric mucosal barrier is influenced by mucus secretion,

acid secretion, and gastric blood flow. The gastric irritation of non-steroidal anti-inflammatory drugs on the gastric mucosa is one of the major disadvantages of their using inflammatory. It is well established that NSAIDs causes inhibition of the synthesis of cytoprotective prostaglandin.

Recently, widespread effort has been launched to identify novel anti-ulcer drugs from natural resources. Ulcer is defined as the erosion in the lining of the stomach or duodenum and is caused by the disruptions of the gastric mucosal defense and repair systems. Ulcer in the stomach is called gastric ulcer and in the duodenum is called duodenal ulcer and together peptic ulcer. In clinical practice, peptic ulcer is one of the most prevalent gastrointestinal disorders, commonly occurs in developed countries.

Treatments available for ulcer is generally non-specific and is usually aimed at reducing the production of gastric acid and re-enforcing gastric mucosal protection such as regular food, adequate rest and avoidance of ulcerogenic agents such as coffee, alcohol and tobacco. The drugs used in the treatment of ulcer include receptor blockers, proton pump inhibitors, drugs affecting the mucosal barrier and act on the central nervous system. Even though a range of drugs are available for the treatment of ulcer, many of these do not fulfill all the requirements and have side effects. Recently, there has been much interest in natural medicines derived from the traditional knowledge of plant pharmacological properties. The present study was undertaken to evaluate the effect of Panai Poo Chooranam on Aspirin induced model of gastro intestinal ulceration.

**ANTI-ULCER EVALUATION**

Aspirin induced gastric ulcer:

Animals were divided into five groups (n = 6) *Panai Poo Chooranam* (250, 500mg/kg) inter peritoneal and control vehicle were administered 30 min. before the administration of aspirin (400mg/kg) per orally. The animals were scarified, after 6 hours following the administration of aspirin, stomachs were removed and 2% formalin was injected into the stomach. The stomach was open along with greater

curvature and immersed in 2% formalin solution. The length of each lesion was measured under the dissecting microscope. The sum of the length (mm) of all lesions for each rat was used in lesion index.

The ulcer score was determined by using a 10 × magnifying hand lens. The scoring of severity of ulceration was as follows:

1 mm (pin point) = 1; 1-2 mm = 2; > 2 mm = 3; > 3 mm = 4. The mean ulcer score was determined by dividing the total ulcer indices in a group by the total number of animals in that group. Ulcer Score = Total ulcer index (UI) in a group/Total number of animals in that group.

***Statistical analysis***

The statistical analysis was carried out using one-way ANOVA followed by Dunnett's multiple comparison test. All the results obtained in the study were compared with the vehicle control group. P values <0.05 were considered statistically significant.

## 4.5. CLINICAL ASSESSMENT

I have selected the herbal drug which proved its anti – ulcer activity pre clinically. *Panai poo chooranam*, a herbal medicine was used for this clinical trial to prove its safety and efficacy against Peptic ulcer disease.

### Objectives

The study was conducted on peptic ulcer patients to assess the "anti-ulcer" activity of ***"Panai poo chooranam"*** clinically, both in-patients and out-patients of both sex and varying age groups.

### Study centre

The clinical study for **PEPTIC ULCER** is carried out in outpatient department and in patient ward of Govt.Siddha medical college hospital and Arignar Anna Indian Hospital, Arumbakkam, Chennai-106.

**Design of the study**

Open clinical trial, phase II B

**Selection**

50 patients from both sexes of various age groups were selected for clinical trial. Among 50 patients 40 patients were treated as out-patients, 10 patients were treated as in-patients. The selection was based on the including and excluding criteria. They were clinically diagnosed on the basis of siddha principles with modern laboratory findings.

**Registration process**

To register a patient, the following documents have been proceeded.

- Copy of required laboratory tests
- Signed patient consent form

Then I verified eligibility and assigned a patient study number, drug dose and registered the patient on the study.

**Criteria selection**

**Including criteria**

- Epigastric pain
- Heart burn
- Regurgitation
- Nausea/vomiting
- Loss of appetite
- Abdominal discomfort

**Excluding criteria**

- Complication of peptic ulcer such as
  - Haemorrhage,
  - Perforation,
  - Gastric outlet obstruction
- Radiating abdominal pain as in pancreatitis, appendicitis
- Acute abdominal colic's
- Cancer of the stomach
- Gall stone and hiatus hernia
- Cirrhosis of liver and jaundice

**Criteria for withdrawal**

Patients were removed from study when any of the criteria listed below applies. In the absence of treatment delays due to adverse events, treatment may continue for 2 cycles or until one of the following criteria applies:

- Disease progression,
- Deterioration of vital signs with cardiac, respiratory, hepatic, renal and CNS changes.
- Intercurrent illness that prevents further administration of treatment,
- Unacceptable adverse event(s),
- Patient decides to withdraw from the study, or
- General or specific changes in the patient's condition render the patient unacceptable for further treatment in the judgment of the investigator.

**Investigation**

For all the cases full clinical data were recorded and they were diagnosed on the basis of siddha principles i.e. *Envagai thrvugal*, *Ezhu udal thathukkal* etc.

All the patients under study were subjected to blood investigations for TC, DC, ESR, and Hb. Blood urea, serum cholesterol and Blood sugar were also investigated.

Urine test for albumin, sugar, deposits and motion test for ova, cysts were done.

The disease *Peptic Ulcer* was confirmed in the patients by means of Endoscope examination, Barium meal examination and clinically.

**Routine examination and assessment**

The full details of history and physical examination of the patients were recorded as per the proforma. The clinical assessment was done initially at the end of 7 days, 14 days and 21 days during treatment and at the end of the 21 days follow up were done. The laboratory investigation and the physiological parameters will be recorded initially and the end of the treatment and at follows up as per the Proforma.

**Administration of the drug:**

| | |
|---|---|
| Form of the medicine | : *Chooranam* |
| Route of Administration | : Enteral |
| Dose | : 1gm |
| *Anubanam* (Vehicle) | : Warm water |
| Times of Administration | : Two times a day; before food |
| Duration | : 7 weeks |

**Diet and medical advice**

**Do's**

- Timely food
- Banana
- Carrots and cabbage juice
- Butter milk
- Should chew every morsel thoroughly
- Meals must be small and frequent

**Dont's**

- Fatty and tough meats
- Fried foods, Sour foods
- Unripe citrus fruits like oranges and sweet lime
- Spicy foods, carbonated drinks
- Strongly flavored veggies like cauliflower, turnip, radish, onion, etc
- Strong tea and coffee
- Alcoholic Beverages
- Intake of steroids and NSAIDS.
- Above all avoid worrying.

**Trial conduct**

This study will be conducted in compliance with the protocol approved by the Institutional Review Board, and according to Good Clinical Practice standards. No deviation from the protocol will be implemented without the prior review and approval of the IRB except where it may be necessary to eliminate an immediate hazard to a research subject. In such case, the deviation will be reported to the IRB as soon as possible.

**Criteria for assessment of response to therapy:**

1) Good Relief : 75% relief in the presenting signs and symptoms marked normality pathological investigation.

2) Satisfactory : 60 – 75% relief signs and symptoms, moderate normality of pathological investigation.

3) Moderate : 50% relief of signs and symptoms, mild changes in pathological investigations.

4) Poor **:** less than 50% relief in symptoms and no significant improvement in laboratory parameters.

Then clinical signs and symptoms like Epigastric pain, Heart burn, regurgitation and distension of abdomen were observed regularly under the supervision of HOD, Lecturers, and Asst.Lecturers.

**Follow up**

Assessment was taken for every three days before treatment and after treatment. During this period clinical assessment and laboratory investigation was carried out.

**Statistical analysis**

The data will be tabulated and analyzed by student‘t’ test.

**Ethical review**

The protocol and amendments were submitted to the Govt siddha medical college, Institutional Ethical Committee (IEC) and got formal approval for conducting the study. The decision of the IEC concerning the conduct of the study was made in writing to the investigator.

All subjects for this study was provided a consent form describing this study and provided sufficient information for subjects to make an informed decision about

their participation in this study. This consent form was submitted with the protocol for review and approval by the IEC. The formal consent of a subject, using the IEC-approved consent form, was obtained before that subject is submitted to any study procedure. This consent form was signed by the subject or legally acceptable surrogate, and the investigator-designated research professional obtaining the consent.

## Table No: 4.5.1

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN OUT PATIENTS DEPT. IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 1. | 2823 | Theivanayagi 60/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 3.6.12 To 15.7.12 | BT | 8700 | 53 | 40 | 7 | 30 | 71 | 9.6 | 88 | 23 | 159 | NIL | NIL | PCS | | Good |
| | | | | | AT | 8700 | 54 | 39 | 7 | 30 | 71 | 9.6 | 88 | 20 | 152 | NIL | NIL | NIL | | |
| 2. | 7268 | Ajith 23/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 4.6.12 To 28.7.12 | BT | 10000 | 64 | 32 | 4 | 2 | 4 | 14.4 | 89 | 21 | 159 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 10000 | 65 | 32 | 4 | 2 | 4 | 14.4 | 89 | 19 | 160 | NIL | NIL | NIL | | |
| 3. | 7276 | Saraswathy 25/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 4.6.12 To 28.7.12 | BT | 8800 | 60 | 37 | 3 | 8 | 12 | 10 | 90 | 20 | 173 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 8800 | 61 | 37 | 2 | 8 | 12 | 10 | 90 | 18 | 170 | NIL | NIL | NIL | | |
| 4. | 4271 | Rangasamy 45/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 5.6.12 To 10.8.12 | BT | 9800 | 62 | 34 | 4 | 3 | 6 | 13 | 92 | 24 | 165 | NIL | NIL | FPC | - | Satisfactory |
| | | | | | AT | 9800 | 61 | 35 | 4 | 3 | 6 | 13 | 92 | 22 | 163 | NIL | NIL | NIL | | |
| 5. | 5167 | Chandra 32/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 6.6.12 To 25.7.12 | BT | 9000 | 58 | 38 | 4 | 26 | 44 | 12 | 94 | 22 | 162 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 9000 | 58 | 38 | 3 | 27 | 43 | 12 | 94 | 22 | 160 | NIL | NIL | NIL | | |

## Table No: 4.5.2

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN OUT PATIENTS DEPT. IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 6. | 1919 | Sumathy 29/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 7.6.12 To 15.7.12 | BT | 9600 | 55 | 41 | 4 | 12 | 20 | 11.5 | 94 | 20 | 150 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 9600 | 54 | 42 | 4 | 12 | 20 | 11.5 | 94 | 21 | 148 | NIL | NIL | NIL | | |
| 7. | 1575 | Kiruba rani 35/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 8.6.12 To 18.7.12 | BT | 9300 | 54 | 40 | 6 | 15 | 22 | 12 | 85 | 23 | 164 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 9300 | 56 | 39 | 5 | 15 | 22 | 12 | 86 | 20 | 163 | NIL | NIL | NIL | | |
| 8. | 1781 | Sakthivel 47/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 9.6.12 To 10.7.12 | BT | 9800 | 59 | 39 | 2 | 4 | 6 | 14 | 95 | 21 | 159 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 9800 | 58 | 40 | 2 | 4 | 6 | 14 | 95 | 19 | 162 | NIL | NIL | NIL | | |
| 9. | 9410 | Prabhu 30/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 12.6.12 To 29.7.12 | BT | 9900 | 57 | 39 | 4 | 6 | 10 | 13 | 99 | 19 | 161 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 9900 | 57 | 39 | 4 | 6 | 10 | 13 | 98 | 18 | 157 | NIL | NIL | NIL | | |
| 10. | 302 | Palaniyammal 36/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 14.6.12 To 10.8.12 | BT | 8800 | 53 | 42 | 5 | 15 | 24 | 11 | 89 | 19 | 190 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 8800 | 54 | 41 | 5 | 15 | 20 | 11.5 | 89 | 20 | 188 | NIL | NIL | NIL | | |

## Table No: 4.5.3

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN OUT PATIENTS DEPT. IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 11. | 126 | Madhaiyan 41/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 16.6.12 To 10.8.12 | BT | 9700 | 60 | 37 | 3 | 4 | 8 | 13 | 90 | 17 | 170 | NIL | NIL | PCS | - | Moderate |
| | | | | | AT | 9700 | 60 | 37 | 3 | 4 | 8 | 13 | 90 | 19 | 173 | NIL | NIL | NIL | | |
| 12. | 7657 | Ayisha 60/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 19.6.12 To 4.8.12 | BT | 8700 | 57 | 38 | 5 | 20 | 45 | 8 | 150 | 21 | 168 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 8700 | 56 | 40 | 4 | 18 | 40 | 9 | 150 | 19 | 162 | NIL | NIL | NIL | | |
| 13. | 9039 | Tarus 47/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 21.6.12 To 30.7.12 | BT | 9000 | 58 | 36 | 6 | 10 | 16 | 10 | 85 | 23 | 171 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 9000 | 57 | 38 | 5 | 10 | 16 | 10 | 84 | 20 | 168 | NIL | NIL | NIL | | |
| 14. | 4216 | Manonmani 29/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 23.6.12 To 29.7.12 | BT | 8900 | 59 | 37 | 4 | 8 | 12 | 12 | 82 | 18 | 162 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 8900 | 58 | 38 | 4 | 10 | 12 | 12 | 84 | 20 | 166 | NIL | NIL | NIL | | |
| 15. | 783 | Sofiya 32/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 27.6.12 To 12.8.12 | BT | 9300 | 61 | 35 | 4 | 10 | 14 | 11 | 98 | 26 | 158 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 9300 | 61 | 35 | 4 | 10 | 14 | 11 | 100 | 28 | 160 | NIL | NIL | NIL | | |

## Table No: 4.5.4

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN OUT PATIENTS DEPT. IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 16. | 784 | Shanthi 51/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 27.6.12 To 23.8.12 | BT | 9000 | 54 | 41 | 5 | 11 | 20 | 10.8 | 100 | 21 | 155 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 9000 | 55 | 42 | 3 | 12 | 20 | 11 | 100 | 19 | 160 | NIL | NIL | FPC | | |
| 17. | 1048 | Rajendran 50/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 28.6.12 To 2.8.12 | BT | 9700 | 60 | 37 | 3 | 8 | 12 | 13 | 94 | 21 | 158 | NIL | NIL | NIL | - | Moderate |
| | | | | | AT | 9700 | 60 | 36 | 4 | 8 | 12 | 13 | 92 | 20 | 156 | NIL | NIL | NIL | | |
| 18. | 3255 | Suresh 32/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 2.7.12 To 25.8.12 | BT | 9800 | 61 | 35 | 4 | 4 | 8 | 14 | 90 | 19 | 160 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 9800 | 62 | 34 | 4 | 4 | 8 | 14 | 90 | 20 | 162 | NIL | NIL | NIL | | |
| 19. | 4027 | Ganapathy 29/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 4.7.12 To 29.8.12 | BT | 9500 | 60 | 38 | 2 | 8 | 10 | 13.5 | 89 | 26 | 165 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 9500 | 61 | 37 | 2 | 8 | 10 | 13.5 | 90 | 26 | 158 | NIL | NIL | NIL | | |
| 20. | 3043 | Selvalakshmi 35/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 7.7.12 To 31.8.12 | BT | 8600 | 55 | 40 | 5 | 22 | 52 | 12 | 89 | 19 | 159 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 8600 | 55 | 39 | 6 | 10 | 20 | 13 | 86 | 22 | 160 | NIL | NIL | FPC | | |

## Table No: 4.5.5

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN OUT PATIENTS DEPT. IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 21. | 3046 | Amaravathi 46/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 9.7.12 To 28.8.12 | BT | 10000 | 60 | 36 | 4 | 8 | 14 | 11.5 | 105 | 20 | 162 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 10000 | 60 | 37 | 3 | 8 | 14 | 11.5 | 106 | 19 | 160 | NIL | NIL | FPC | | |
| 22. | 3017 | Srinivasan 30/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 10.7.12 To 5.9.12 | BT | 9800 | 62 | 34 | 4 | 4 | 8 | 13 | 101 | 18 | 157 | NIL | NIL | PCS | - | Moderate |
| | | | | | AT | 9800 | 63 | 34 | 3 | 4 | 8 | 13 | 98 | 20 | 160 | NIL | NIL | NIL | | |
| 23. | 4581 | Sumathy 31/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 12.7.12 To 21.8.12 | BT | 8000 | 55 | 41 | 4 | 45 | 80 | 9 | 93 | 21 | 162 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 8200 | 56 | 40 | 4 | 40 | 70 | 10 | 90 | 20 | 160 | NIL | NIL | NIL | | |
| 24. | 4032 | Chitra 37/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 14.7.12 To 25.8.12 | BT | 8500 | 58 | 37 | 5 | 20 | 30 | 10 | 89 | 22 | 168 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 8500 | 58 | 37 | 5 | 20 | 30 | 10 | 91 | 20 | 161 | NIL | NIL | FPC | | |
| 25. | 5967 | Visalakshi 53/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 18.7.12 To 28.8.12 | BT | 7600 | 53 | 41 | 6 | 30 | 56 | 8 | 100 | 20 | 153 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 8000 | 53 | 42 | 5 | 20 | 40 | 10 | 96 | 18 | 155 | NIL | NIL | NIL | | |

## Table No: 4.5.6

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN OUT PATIENTS DEPT. IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 26. | 5811 | Sinthamani 39/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 20.7.12 To 30.8.12 | BT | 9600 | 58 | 38 | 4 | 10 | 14 | 12 | 90 | 24 | 159 | NIL | NIL | FPC | - | Satisfactory |
| | | | | | AT | 9600 | 59 | 38 | 3 | 10 | 14 | 12 | 88 | 22 | 160 | NIL | NIL | NIL | | |
| 27. | 4741 | Mariappan 40/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 21.7.12 To 7.9.12 | BT | 9800 | 60 | 35 | 5 | 14 | 20 | 13 | 98 | 20 | 162 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 9800 | 61 | 35 | 4 | 14 | 20 | 13.5 | 96 | 18 | 161 | NIL | NIL | NIL | | |
| 28. | 8263 | Saraswathy 40/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 26.7.12 To 12.12.11 | BT | 9200 | 58 | 37 | 5 | 8 | 12 | 12 | 85 | 20 | 170 | NIL | NIL | NIL | | Good |
| | | | | | AT | 9200 | 59 | 36 | 5 | 8 | 12 | 12 | 88 | 18 | 168 | NIL | NIL | NIL | - | |
| 29. | 8606 | Kumar 51/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 28.7.12 To 10.9.12 | BT | 10600 | 64 | 31 | 5 | 10 | 18 | 12.6 | 96 | 19 | 167 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 10600 | 65 | 31 | 4 | 10 | 14 | 13 | 98 | 20 | 168 | NIL | NIL | FPC | | |
| 30. | 361 | Balakrishnan 45/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 3.8.12 To 28.9.12 | BT | 9400 | 62 | 35 | 3 | 4 | 8 | 14 | 108 | 20 | 158 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 9400 | 64 | 34 | 2 | 4 | 8 | 14 | 106 | 20 | 160 | NIL | NIL | NIL | | |

## Table No: 4.5.7

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN OUT PATIENTS DEPT. IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 31. | 3278 | Mamitha banu 44/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 16.8.12 To 29.9.12 | BT | 9400 | 60 | 36 | 4 | 20 | 36 | 11 | 98 | 19 | 157 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 9400 | 59 | 38 | 3 | 20 | 36 | 11 | 100 | 16 | 155 | NIL | NIL | NIL | | |
| 32. | 5548 | Kumar 28/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 27.8.12 To 20.9.12 | BT | 10100 | 62 | 35 | 3 | 6 | 12 | 14 | 90 | 22 | 167 | NIL | NIL | PCS | - | Poor |
| | | | | | AT | 10100 | 62 | 36 | 2 | 6 | 12 | 14 | 90 | 20 | 163 | NIL | NIL | NIL | | |
| 33. | 5549 | Shanthi 35/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 27.8.12 To 6.10.12 | BT | 9600 | 57 | 40 | 3 | 18 | 25 | 9 | 100 | 23 | 157 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 9600 | 56 | 41 | 3 | 18 | 25 | 9 | 97 | 22 | 162 | NIL | NIL | FPC | | |
| 34. | 7443 | Rani 44/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 4.9.12 To 14.10.12 | BT | 9800 | 59 | 37 | 4 | 10 | 26 | 10.8 | 100 | 16 | 150 | NIL | NIL | NIL | - | Satisfactory |
| | | | | | AT | 9800 | 59 | 38 | 3 | 10 | 24 | 11 | 98 | 17 | 154 | NIL | NIL | NIL | | |
| 35. | 2107 | Balamurali 36/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 24.9.12 To 27.10.12 | BT | 10000 | 60 | 35 | 5 | 10 | 16 | 13 | 118 | 22 | 159 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 10000 | 60 | 36 | 4 | 10 | 16 | 13 | 114 | 20 | 158 | NIL | NIL | FPC | | |

## Table No: 4.5.8

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN OUT PATIENTS DEPT. IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 36. | 2639 | Shajahan 50/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 26.9.12 To 29.10.12 | BT | 10700 | 66 | 29 | 5 | 7 | 12 | 14 | 108 | 23 | 158 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 10700 | 66 | 31 | 3 | 7 | 12 | 14 | 104 | 20 | 155 | NIL | NIL | NIL | | |
| 37. | 6363 | Shakilabegam 26/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 11.10.12 To 18.11.12 | BT | 9600 | 62 | 34 | 4 | 12 | 20 | 12 | 92 | 19 | 158 | NIL | NIL | PCS | - | Moderate |
| | | | | | AT | 9600 | 62 | 35 | 3 | 12 | 20 | 12 | 92 | 20 | 160 | NIL | NIL | FPC | | |
| 38. | 319 | Meenakshi sundaram 50/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 31.10.12 To 29.11.12 | BT | 10500 | 64 | 32 | 4 | 6 | 10 | 14 | 120 | 21 | 162 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 10500 | 64 | 34 | 2 | 6 | 10 | 14 | 118 | 18 | 163 | NIL | NIL | FPC | | |
| 39. | 2543 | Prabhavathi 37/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 10.11.12 To 12.12.12 | BT | 9300 | 60 | 34 | 6 | 10 | 24 | 11 | 88 | 22 | 157 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 9300 | 62 | 33 | 5 | 10 | 24 | 11 | 87 | 20 | 160 | NIL | NIL | NIL | | |
| 40. | 1925 | Ganthamani 35/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 7.11.12 To 15.12.12 | BT | 8700 | 60 | 36 | 4 | 25 | 40 | 9 | 98 | 22 | 159 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 8500 | 60 | 37 | 3 | 20 | 35 | 10 | 95 | 20 | 158 | NIL | NIL | NIL | | |

## Table No: 4.5.9

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN, IN-PATIENTS DEPT.IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | IP. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 1. | 663/ 6046 | Thaiyal nayagi 55/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 12.6.12 To 28.6.12 | BT | 8700 | 53 | 40 | 7 | 30 | 71 | 9.6 | 88 | 23 | 156 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 8700 | 54 | 41 | 5 | 25 | 40 | 11 | 90 | 22 | 154 | NIL | NIL | NIL | | |
| 2. | 1052/ 6013 | Sinnaponnu 50/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 18.7.12 To 4.8.12 | BT | 9200 | 57 | 40 | 3 | 15 | 28 | 12 | 108 | 23 | 159 | NIL | NIL | PCS | - | Poor |
| | | | | | AT | 9200 | 57 | 41 | 2 | 15 | 25 | 12 | 104 | 20 | 152 | NIL | NIL | NIL | | |
| 3. | 1059/ 7509 | Krishnaprasanth 62/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 24.7.12 To 11.9.12 | BT | 10200 | 62 | 35 | 3 | 18 | 26 | 11.5 | 106 | 20 | 171 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 10000 | 63 | 34 | 3 | 15 | 23 | 12 | 102 | 19 | 169 | NIL | NIL | NIL | | |
| 4. | 1115/ 7963 | Lakshmi 45/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 25.7.12 To 5.8.12 | BT | 9000 | 52 | 43 | 5 | 18 | 28 | 11 | 100 | 23 | 164 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 9100 | 60 | 36 | 4 | 18 | 28 | 11 | 102 | 20 | 162 | NIL | NIL | NIL | | |
| 5. | 1132/ 8234 | Aarumugathammal 60/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 26.7.12 To 8.8.12 | BT | 8800 | 54 | 40 | 6 | 20 | 35 | 10 | 106 | 21 | 163 | NIL | NIL | NIL | - | Satisfactory |
| | | | | | AT | 8900 | 55 | 40 | 5 | 20 | 35 | 10 | 106 | 23 | 160 | NIL | NIL | NIL | | |

## Table No: 4.5.10

## CLINICAL STUDY ON *PANAI POO CHOORANAM* IN, IN-PATIENTS DEPT.IN THE MANAGEMENT OF PEPTIC ULCER

| SI NO | IP. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | Blood CL | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 6. | 1170/ 9601 | Krishnaveni 45/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 1.8.12 To 18.9.12 | BT | 8100 | 56 | 38 | 6 | 18 | 24 | 10 | 96 | 22 | 160 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 8200 | 56 | 39 | 5 | 16 | 22 | 11 | 92 | 20 | 158 | NIL | NIL | NIL | | |
| 7. | 1287/ 3252 | Aarumugam 36/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 16.8.12 To 30.8.12 | BT | 10200 | 63 | 34 | 3 | 10 | 20 | 12 | 106 | 21 | 159 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 10200 | 63 | 33 | 4 | 10 | 14 | 12 | 102 | 20 | 154 | NIL | NIL | NIL | | |
| 8. | 1376/ 6161 | Parvin 54/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 29.8.12 To 7.9.12 | BT | 9700 | 59 | 36 | 5 | 15 | 28 | 11 | 89 | 20 | 168 | NIL | NIL | FPC | - | Moderate |
| | | | | | AT | 9700 | 60 | 37 | 3 | 15 | 25 | 11.5 | 90 | 18 | 164 | NIL | NIL | NIL | | |
| 9. | 1403/ 6882 | Irsan 47/Male | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 1.9.12 To 19.9.12 | BT | 9800 | 62 | 35 | 3 | 6 | 12 | 13 | 102 | 23 | 165 | NIL | NIL | FPC | - | Good |
| | | | | | AT | 9900 | 62 | 36 | 2 | 6 | 12 | 13 | 98 | 20 | 159 | NIL | NIL | NIL | | |
| 10. | 63/ 1884 | Gowri 47/Female | Epigastric pain, Heart burn, Regurgitation, vomiting, Abdominal Distension | 24.9.12 To 3.10.12 | BT | 8600 | 56 | 38 | 6 | 16 | 22 | 10.5 | 94 | 21 | 163 | NIL | NIL | NIL | - | Poor |
| | | | | | AT | 8500 | 57 | 38 | 5 | 14 | 20 | 11 | 92 | 19 | 158 | NIL | NIL | NIL | | |

## 4.5. CLINICAL ASSESSMENT

**Age wise distribution**

**Table No: 4.5.11**

| SL. NO | AGE IN YEARS | NO. OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | 20-30 | 9 | 18 |
| 2 | 31-40 | 16 | 32 |
| 3 | 41-50 | 14 | 28 |
| 4 | 51-60 | 10 | 20 |
| 5 | 61-70 | 1 | 2 |
| TOTAL | | 50 | 100 |

**Inference**

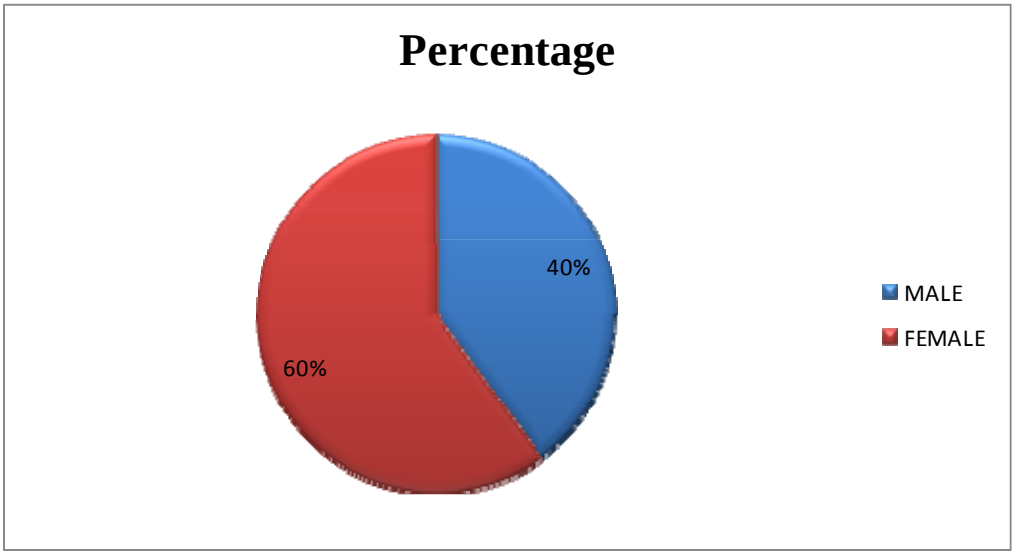
Among 50 patients,

- 9 patients belongs to the age group of 20-30 years
- 16 patients belongs to the age group of 31-40 years
- 14 patients belongs to the age group of 41-50 years
- 10 patients belongs to the age group of 51-60 years
- 1 patient belongs to the age group of 61-70 years

**Sex distribution**

**Table No: 4.5.12**

| SL. NO | SEX | NO. OF PATIENTS | PERCENTAGE |
|---|---|---|---|
| 1 | Male | 20 | 40 |
| 2 | Female | 30 | 60 |
| **TOTAL** | | **50** | **100** |




**Inference:**

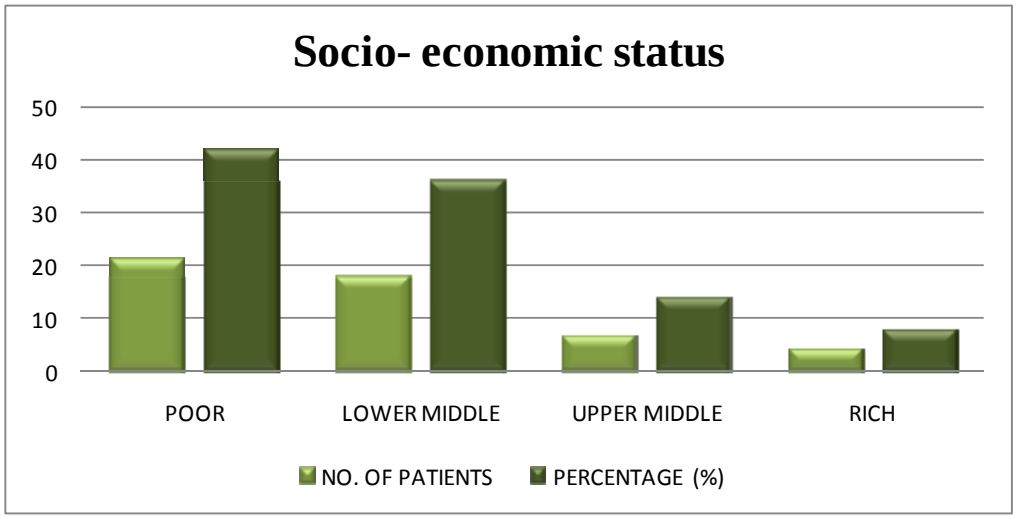
Among 50 patients,

- 20 patients were male
- 30 patients were female

**Socio-economic status**

**Table No: 4.5.13**

| SL. NO | SOCIO – ECONOMIC STATUS | NO. OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | Poor | 21 | 42 |
| 2 | Lower middle | 18 | 36 |
| 3 | Upper middle | 7 | 14 |
| 4 | Rich | 4 | 8 |
| TOTAL | | 50 | 100 |




**Inference:**

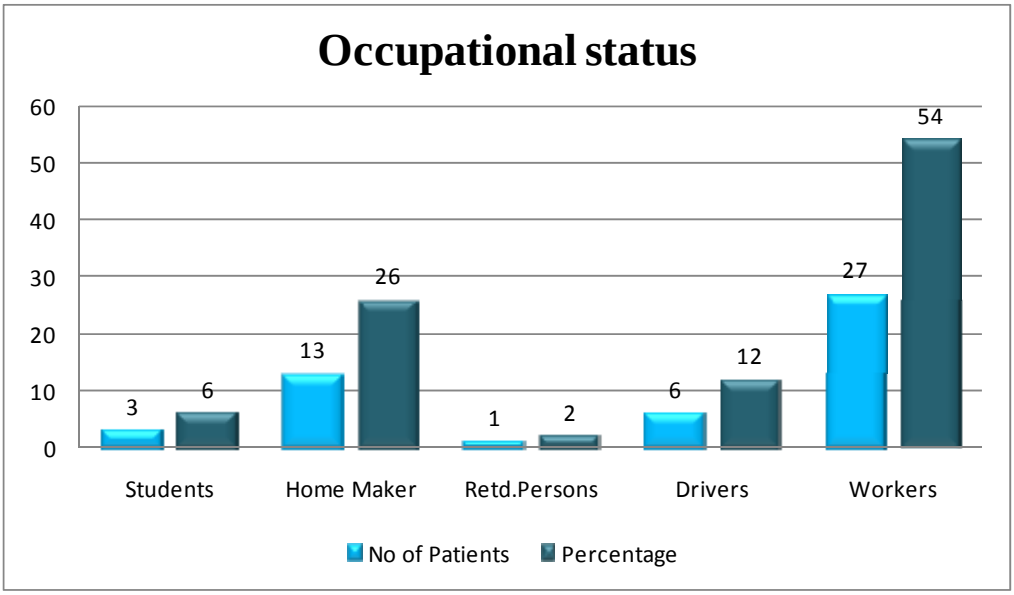
Among 50 patients,

- 21 patients were poor.
- 18 patients were lower-middle.
- 7 patients were upper middle.
- 4 patients were rich.

**Occupational status**

**Table No: 4.5.14**

| SL. NO | OCCUPATION | NO. OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | Students | 3 | 6 |
| 2 | Home makers | 13 | 26 |
| 3 | Retired persons | 1 | 2 |
| 4 | Drivers | 6 | 12 |
| 5 | Workers | 27 | 54 |
| TOTAL | | 50 | 100 |




**Inference**

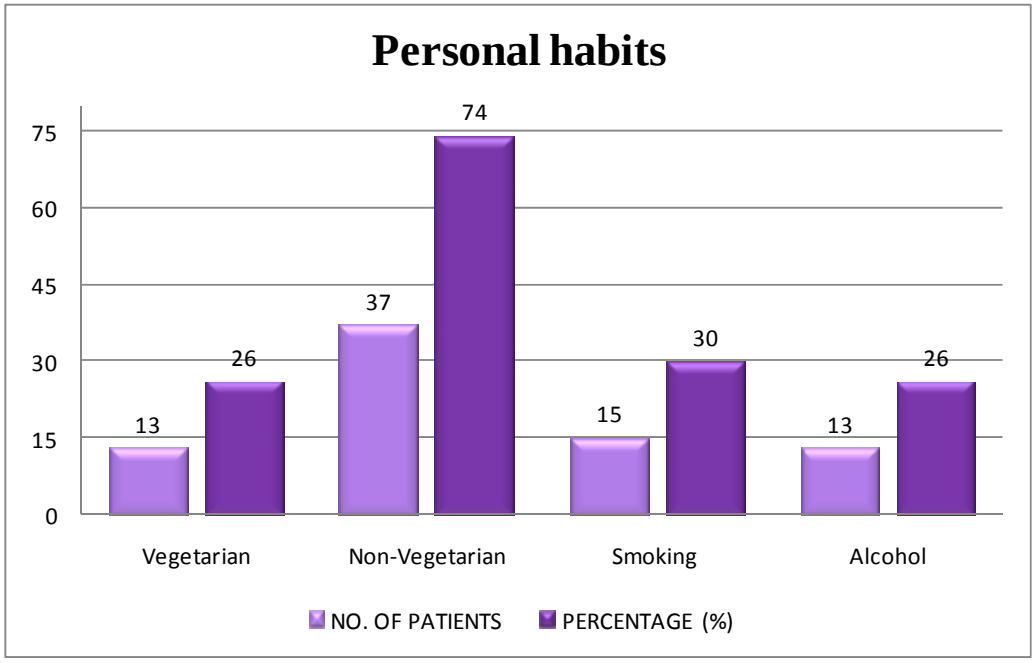
Among 50 patients,

- 3 (6%) patients were Students.
- 13 (26%) patients were Home makers.
- 1 (2%) patients were Retired persons.
- 6 (12%) patients were Drivers.
- 27 (54%) patients were Workers.

**Personal habits**

**Table No: 4.5.15**

| SL. NO | PERSONAL HABITS | NO. OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | Vegetarian | 11 | 22 |
| 2 | Non-vegetarian | 39 | 78 |
| 3 | Smoking | 15 | 30 |
| 4 | Alcohol | 13 | 26 |



# 5. RESULTS AND DISCUSSION

*Panai poo chooranam* was taken for the treatment of Peptic ulcer disease in reference with *Agathiyar gunavagdam.*

There are various studies done in the trial drug *Panai poo chooranam.* The study includes literary collections, Pharmacognostic study, physico and Phyto chemical analysis, toxicological study, pharmacological study and clinical study.

Literature collections about the drug indicate the efficiency of the drug in the treatment of Peptic ulcer.

Botanical aspect deals with the identification, description, cultivation and ethno medicinal importance of the plant.

**Gunapadam aspect** expressed strongly that the drug possess good Anti ulcer property.

### 4.2.1. PHARMACOGNOSTIC ASPECT OF *Borassus flabellifer* Linn.

**Structure of the Perianth**

The perianth is thick and woody. The central part is thick and the marginal part becomes thin. In cross sectional view the perianth exhibits outer epidermis, middle ground tissue with numerous scattered vascular bundles and inner epidermis( Fig4.2.1.2 ). The outer epidermis is stomatiferous with deeply sunken stomata. ( Fig4.2.1.2 and 4.2.1.3). The outer epidermal cells are radially rectangular, thin walled with prominent article.(Fig 4.2.1.3). The epitermal cells are 20µm thick. The inner epidermis is comparatively thin and the cells are spindle shaped and the article is prominent. The ground tissue of the perianth is parenchymatous and most of the cells posses dense accumulation of darkly stained tannin. Diffusely distributed in the ground tissue there are numerous vascular strands. The vascular strands are of 2 types some of the strands consists of only fibers and no vessels and phloem elements are seen within bundle sheath fibers. Fibers sheath may enclose entirely, the vascular elements or the fibers may occur as caps on the outer and inner parts of the strands. (Fig 4.2.1.1and 4.2.1.5).

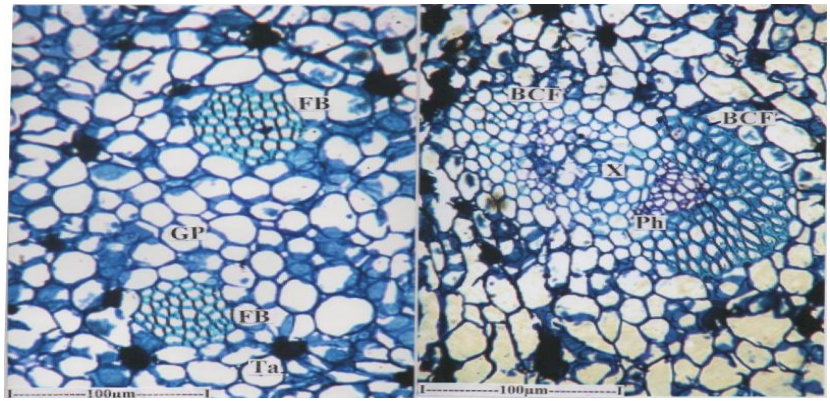
The vascular strands having both phloem and xylem are collateral with outer phloem and inner xylem. The phloem occurs as thick, semi circular strand and the phloem elements are narrow and angular. The inner xylem strand diffusely distributed wide and thick walled vessels. No xylem parenchyma is evident. The fiber strands are mostly distributed along the outer and inner zones. The vascular strands are located in the middle portion of the perianth (Fig 4.2.1.2 and 4.2.1.3).

**Structure of the Ovary**

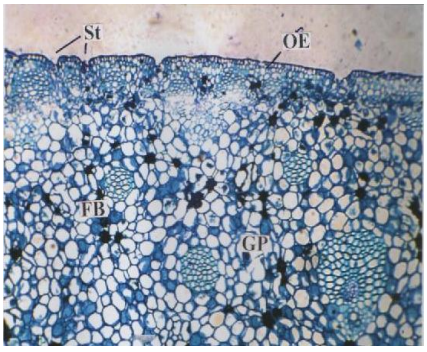
The ovary is tricarpellary syncarpous and 3 chambered (Fig 3.1). The carpels are elliptical and have their walled vesicles, attached all along the carpel wall. The ground tissue is aerenchymatous with several wide airspace distributed diffusely, within the tissue of the ovary. The ovary tissue also contains large number of fiber bundles and vascular bundles (Fig 4.2.1.6, 4.2.1.8 & 4.2.1.10). The epidermal layer of the ovary consists of radially elongated thick walled cylindrical cells with thick cuticle. The fiber bundles occurs throughout the ovary (pericarp) they are circular comprising thick walled, lignified fibers. The fiber bundles are 70µm_in diameter . The vascular bundles are either circular , elliptical or ovate in outline. The bundles have central core of collateral, xylem and phloem elements. The fibers occur either entirely enclosing the vascular elements or the fibers may occur as thick arcs on phloem side and xylem side.(Fig 4.2.1.8, 4.2.1.7). The xylem elements of the vascular bundles occur in cluster. They are wide, regular and thick walled. Phloem elements are seen in shallow arc. The phloem elements are wide, regular and thick walled. ( Fig 4.2.1.11 ) The xylem elements are upto 30µm in dia meter.

**T.S. of Perianth**

**Figure No: 4.2.1.1 Perianth outer part and inner part**

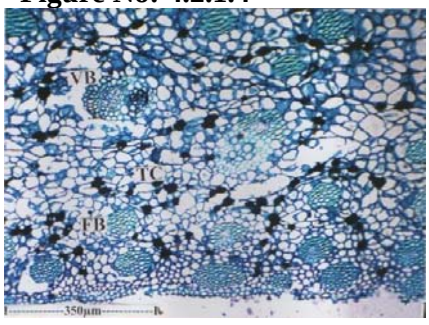



**Perianth outer part:**
**Figure No: 4.2.1.2**

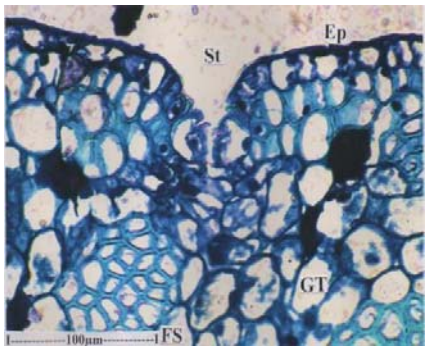



**Perianth inner part**

**Figure No: 4.2.1.4**

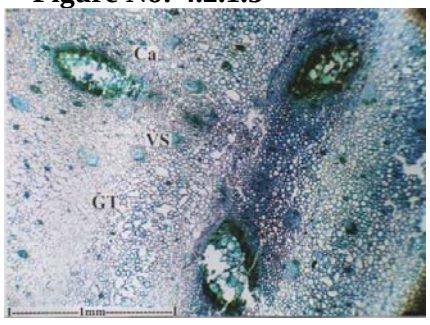



**Perianth outer ep**
**Figure No: 4.2.1.3**

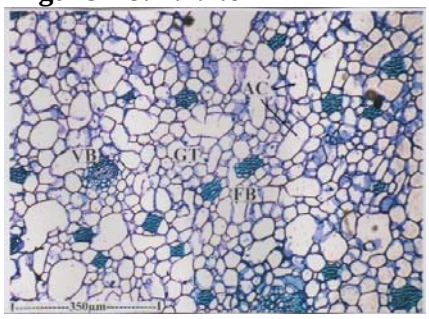



**Ovary central part showing three carpels**

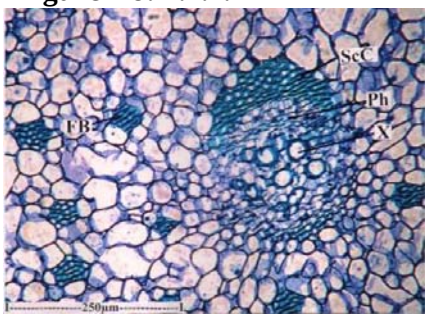
**Figure No: 4.2.1.5**



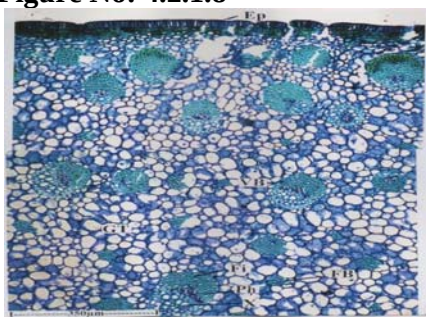
**Ovary air chambers**
**Figure No: 4.2.1.6**



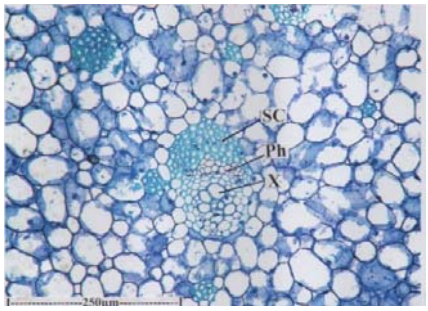

**Ovary VB and central portion**
**Figure No: 4.2.1.7**



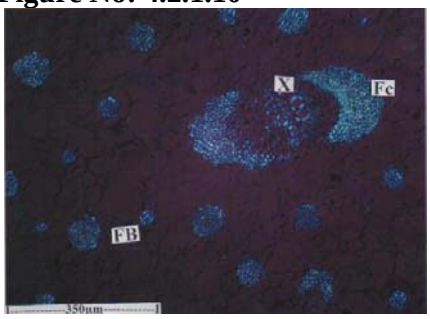

**Ovule Sec 1 and 2**
**Figure No: 4.2.1.8**



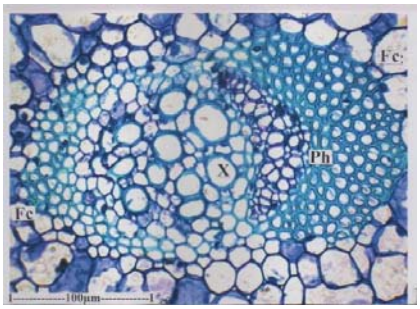

**Ovule VB outer portion**
**Figure No: 4.2.1.9**




**Ovule VB**
**Figure No: 4.2.1.10**




**Ovule single VB enlarged**
**Figure No: 4.2.1.11**



## 4.2.2. PHYSICO-CHEMICAL ANALYSIS

**Table No: 4.2.2.1**

| S.No | Parameter | Mean Value |
|---|---|---|
| 1. | Loss on Drying at 105°C | 5.975 % |
| 2. | Total Ash | 6.048 % |
| 3. | Acid insoluble Ash | 0.473 % |
| 4. | Water Soluble Extractive | 10.0 % |
| 5. | Alcohol Soluble Extractive | 10.15% |
| 6. | pH | 6.6 |

**TLC RESULT OF *PANAI POO CHOORANAM***

**Table No: 4.2.2.2**

| Sl.No | After Dipping in Vanillin-Sulphuric acid | |
|---|---|---|
| | Rf value | Colour of the spot |
| 1 | 0.26 | Grey |
| 2 | 0.36 | Grey |
| 3 | 0.47 | Purple |
| 4 | 0.51 | Purple |
| 5 | 0.67 | Purple |
| 6 | 0.74 | Grey |
| 7 | 0.81 | Blue |

After spray with visualizing agent

**Figure No: 4.2.2.2 TLC result of *Borassus flabellifer***

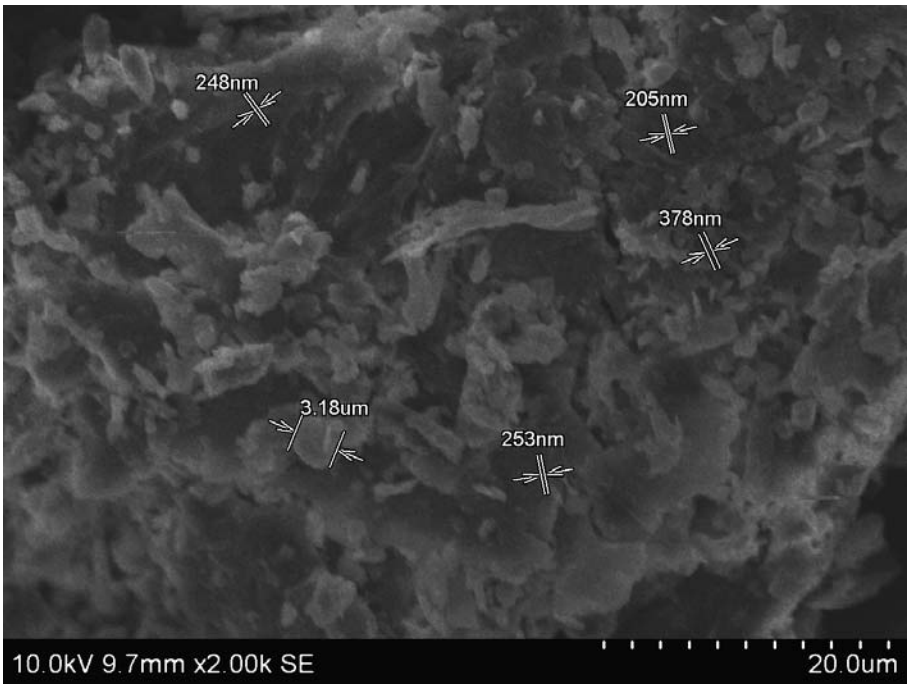
Physico chemical analysis, The analytical parameters like total Ash value, Acid insoluble ash value, Loss on drying values are helping us to interpret the digestion and solubility capacity of the crude extract. As per the result the tested sample contains good percentage of solubility as well as digestive capacity. Gastric juice is highly acidic with pH of 0.9 to 1.2. It is due to the presence of hydrochloric acid. The pH of ***Panai poo chooranam*** is **6.6**. So it neutralizes the acidic pH of gastric juice.

**4.2.2.3 FTIR result of *Panai poo chooranam:***

| PEAK | FUNCTIONAL GROUP |
|---|---|
| 3403$cm^{-1}$ | Amine N-H stretch, Alcohol/ phenol O-H stretch |
| 2924 $cm^{-1}$ | Carboxylic acid O-H stretch |
| 2128 $cm^{-1}$ | Alkynyl C=C stretch |
| 1617 $cm^{-1}$ | C-C with C=O |
| 1521 $cm^{-1}$ | Aromatic C=C bending |
| 1376 $cm^{-1}$ | Alkyl methyl |
| 1062 $cm^{-1}$ | Primary alcohol |
| 612 $cm^{-1}$ | Bromo alkanes |

### 4.2.2.4 SEM result of *Panai poo chooranam*

Figure No: 4.2.4




Result showed particle sizes are: micro particles and nano particles.

Hence Panai poo chooranam is biologically produced micro and nano particles to enhance fast pharmacological action in target site. They are readily absorbable, adaptable and assimilate in the body without producing any adverse effects at therapeutic doses.

### 4.2.5 Qualitative phytochemical analysis:

**Results**: **Phytochemical analysis of** *Borassus flabellifer*

**Table no: 4.2.5**

| **Qualitative Phytochemical Tests** | | |
|---|---|---|
| 1. | Alkaloids | Negative |
| 2. | Triterpenes | Positive |
| 3. | Tannin | Positive |
| 4. | Flavonoids | Negative |
| 5. | Steroids | Positive |
| 6. | Glycoside | Positive |
| 7. | Saponin | Positive |
| 8. | Phenol | Positive |

From the test results Triterpenes, Tannins, Steroids, Glycosides, Saponins, and Phenol were revealed to be present.

Alkaloids and Flavonoids were reported to be absent.

Recent reports and extensive literature review indicated that tannins showed cytoprotective action by increasing mucosal content of prostaglandis and mucous in gastric mucosa

(Dandagi 2008 )

Tannins inhibit gastric mucosal injury by scavenging the stress generated oxygen metabolites.

(Shetty et al – 2008)

Tannin may prevent ulcer development due to their protein precipitating and vasoconstricting effects. Their astringent action can helps precipitating micro proteins on the ulcer site, there by forming an imperious layer over the lining that hinders gut secretions and protects the underlying mucosa from toxins and other irritants.

( Aguwa et al 1988 and Nwafor et al 1996 )

**Disscussion:**

**Tannin** which is present in *Panai poo chooranam* may be responsible for its anti ulcer activity.

**4.2.3. Chemical analysis of *panai poo chooranam*:**

**Results**

The Chemical analysis of *Panai poo Chooranam* showed the following chemicals,

Presence of reducing sugar, starch, proteins, amino acid, albumin, and chloride.

**4.3. Toxicity study**

**Table** No: 4.3 **Dose finding experiment and its behavioral Signs of Toxicity**

| No | Dose mg/kg | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 2000 | + | + | - | + | + | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| 2 | 5000 | + | + | - | + | + | + | - | - | - | - | - | - | - | + | - | - | - | - | - | - |

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

**4.4. PHARMACOLOGICAL STUDY**

**Antiulcer property of *panai poo chooranam***

**Results and discussion**

The examination of acute toxicity carried out on mice indicated that *Panai poo chooranam* has no toxicity when administered orally (up to 5g/kg). Administration of aspirin to fasted rats resulted in severe gastric damage visible from the outside of the

stomach as thick reddish-black lines. After opening, the gastric lesions were found in the mucosa and consisted of elongated bands, 1–8mm long, usually parallel to the long axis of the stomach. They were located mostly in the corpus.

The pathophysiology of ulcers is due to an imbalance between aggressive factors and local mucosal defensive factors (mucus bicarbonate, blood flow and prostaglandins). Prostaglandins are known to play an important role in maintaining mucosal integrity. An Increase in certain endogenous prostaglandins can enhance gastric mucosal resistance to ulcerogenic agents. The mechanisms involved in prostaglandin action are multiple, including stimulation of mucus and bicarbonate output, gastric mucosal blood flow, decreasing gastric motility, increasing the release of endogenous mediators of gastric injury vasoactive amines and leucotrienes and stimulation of cellular growth and repair. The integrity of the gastroduodenal mucosa is maintained through a hemostatic balance between these aggressive and defensive factors. The major cause of gastric ulcer is the chronic use of NSAIDs. Therapeutic and adverse effects of NSAIDs have been attributed to the ability of these drugs to inhibit the action of cyclooxygenase (COX). COX is responsible for the synthesis of prostaglandins that normally inhibit acid secretion, as well as having a protective effect on the gastric mucosa.

In conclusion, *Panai Poo Chooranam* significantly inhibits the occurrence of lesions in stomach but exact mechanisms are not clearly understood. Further studies using more specific methods are required to explore the compounds responsible for the protective effect, and the mechanism of this activity. Ulcer has long been recognized as one of the most important gastrointestinal problem. From this study, it is clear that *Panai Poo Chooranam* have significant anti-ulcer activity in animal models when compared with that of reference drug. The *Panai Poo Chooranam* is non-toxic even at relatively high concentrations. The anti-ulcer activity is probably due to the presence of active principles like tannin.

**Effect of Panai Poo Chooranam on ulcer index**

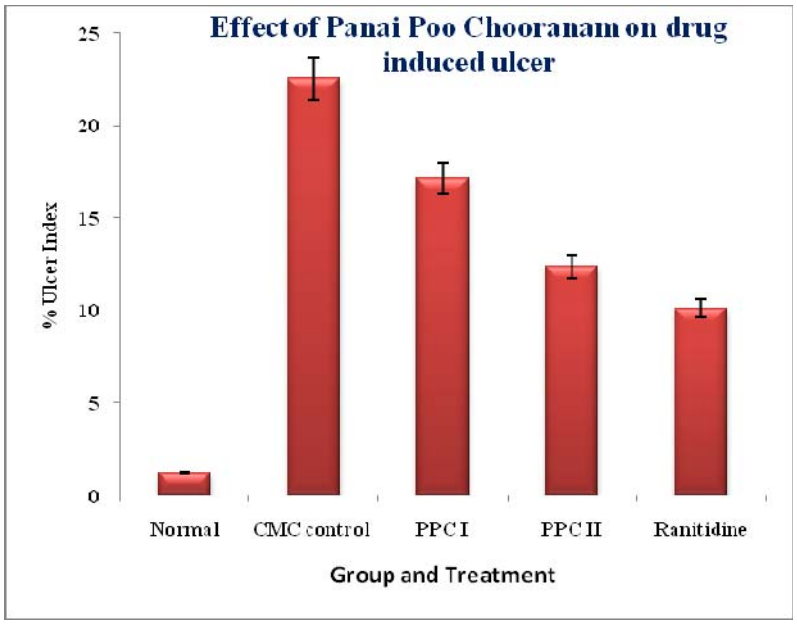
**Table No: 4.4**

| Groups | Ulcer index |
|---|---|
| Normal | 1.22±0.05** |
| CMC control | 22.56 ± 0.28 |
| PPC (250mg/kg) | 17.14 ± 0.21** |
| PPC (500mg/kg) | 12.36 ± 0.25** |
| Ranitidine (60mg/kg) | 10.12 ± 0.14** |

*P values <0.05 as compared to control;

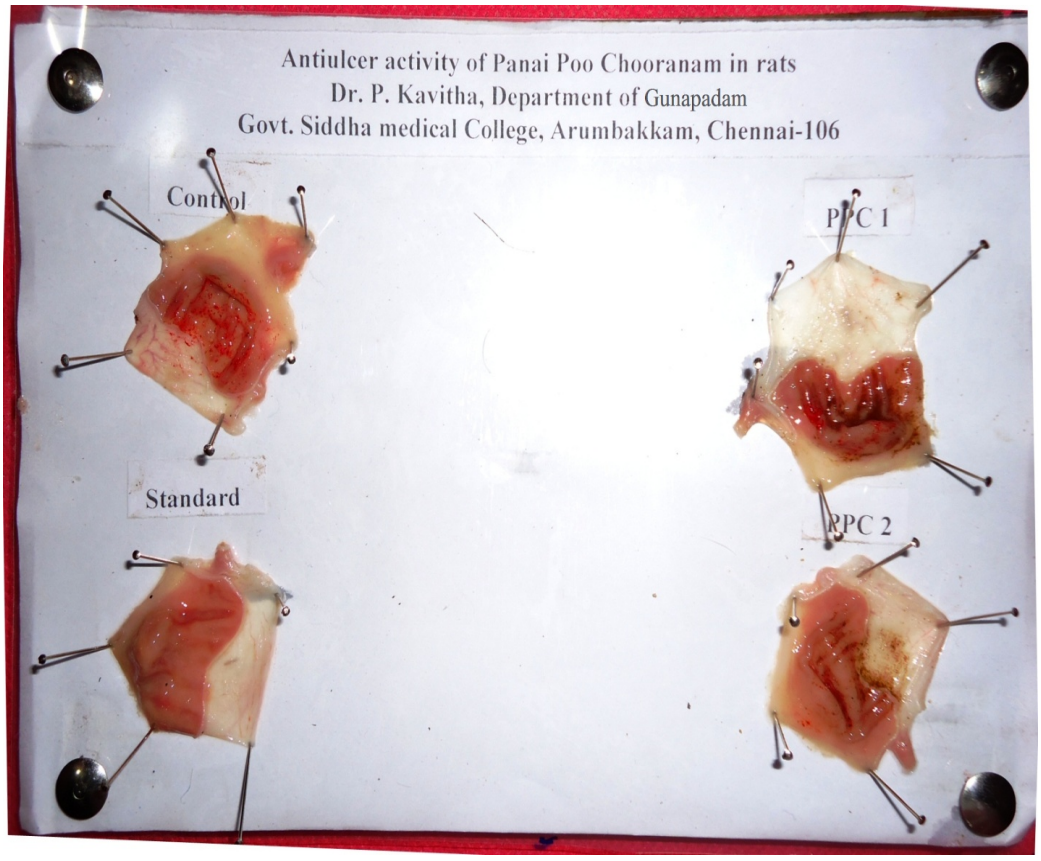
Values are the mean ± S.E.M. of six rats/treatment.

Significance *p <0.05, **p<0.01 Vs Control.



**Anti ulcer activity of *panai poo chooranam***

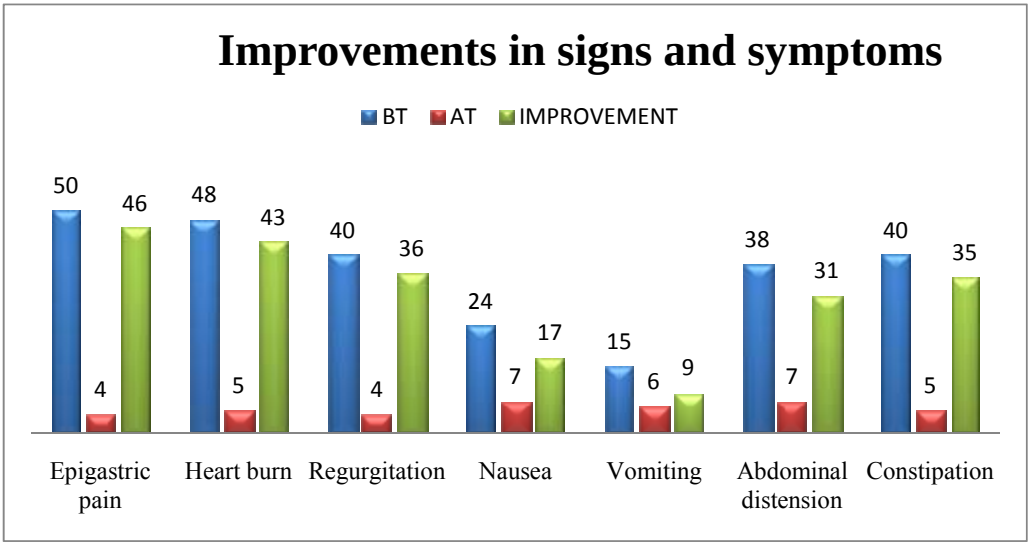
**Figure No: 4.4**



**Clinical assessment- Improvements in signs and symptoms**

**Table No: 4.5.16**

| SL. NO | SIGNS AND SYMPTOMS | NO. OF PATIENTS | | | |
|---|---|---|---|---|---|
| | | BT | AT | IMP. | IMPROVEMENT (%) |
| 1 | Epigastric pain | 50 | 4 | 46 | 92 |
| 2 | Heart burn | 48 | 5 | 43 | 89.5 |
| 3 | Regurgitation | 40 | 4 | 36 | 90 |
| 4 | Nausea | 24 | 7 | 17 | 71 |
| 6 | Vomiting | 15 | 6 | 9 | 60 |
| 7 | Abdominal distension | 38 | 7 | 31 | 81.5 |
| 8 | Constipation | 40 | 5 | 35 | 87.5 |




**Inference:**

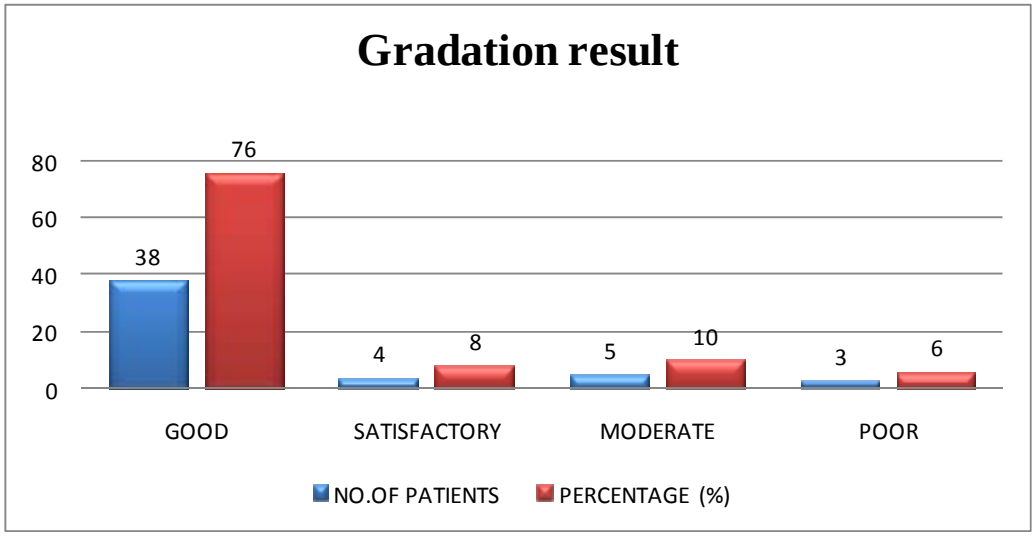
Among 50 patients,

- 46 out of 50 patients were relieved from Epigastric pain.
- 43 out of 48 patients were relieved from heart burn.
- 36 out of 40 patients were relieved from regurgitation.
- 17 out of 24 patients were relieved from nausea.
- 9 out of 15 patients were relieved from vomiting.
- 31 out of 38 patients were relieved from abdominal distension.
- 35 out of 40 patients were relieved from Constipation

**Gradation result**

**Table No: 4.5.17**

| SL. NO | LEVEL OF IMPROVEMENT | NO.OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | Good | 38 | 76 |
| 2 | Satisfactory | 4 | 8 |
| 3 | Moderate | 5 | 10 |
| 4 | Poor | 3 | 6 |
| TOTAL | | 50 | 100 |




**Clinical study**

50 patients of both sexes were selected. Among the 50 patients, 40 patients were treated as out- patients in the Post graduate department of Gunapadam, Govt.Siddha medical college hospital and Govt. Arignar Anna hospital, Arumbakkam, Chennai- 106. 10 patients were treated as in - patients.

The patients were observed regularly.

The trial drug *Panai poo chooranam* was given to the patients at the dose of 1 gm twice a day with warm water before meals. On administration of *Panai poo chooranam* 1 gm twice a day for 7 weeks should significant anti ulcer activity. Warm water which was used as vehicle also has ulcer healing property as per classical siddha literature.

Among 50 patients, 46 out of 50 patients were relieved from Epigastric pain. 43 out of 48 patients were relieved from heart burn. 36 out of 40 patients were relieved from regurgitation. 17 out of 24 patients were relieved from nausea. 9 out of 15 patients were relieved from vomiting. 31 out of 38 patients were relieved from abdominal distension. 35 out of 40 patients were relieved from constipation.

The results revealed that the drug possess 76% good relief, 8% satisfactory relief, 10% moderate relief mild relief, 6% cases there was no improvement.

## STATISTICAL ANALYSIS

### Descriptive statistical for improvement of signs & symptoms in *"Peptic ulcer"*

**Paired *t* test results:**

**P value and statistical significance**

The two-tailed P value is less than 0.0001

By conventional criteria, this difference is considered to be extremely statistically significant.

**Confidence interval**

The mean of Group One minus Group Two equals 5.43

95% confidence interval of this difference: From 4.25 to 6.61

**Intermediate values used in calculations**

t = 11.2877

df = 6

standard error of difference = 0.481

**Result**

**Table No: 5**

| | **Mean** | **S.D** | **S.E.M** |
|---|---|---|---|
| Before treatment | 36.43 | 12.65 | 4.78 |
| After treatment | 31.00 | 13.48 | 5.09 |

**Discussion:**

Improvements of signs and symptoms by statistical analysis shows the two tailed "p" value equals 0.0001, by conventional criteria, this difference is considered to be extremely statistically significant. From the above results $p < 0.05$, it shows the improvement in the subjective parameters produced by *Panai poo chooranam* statistically significant.

# CONCLUSION

The trial drug *Panai poo chooranam* ( *Borassus flabellifer* Linn ) was selected from the classical siddha text *Gunapadam-Mooligai vagupu, Agathiyar gunavaakadam,* for the evaluation of safety and efficacy in the management of Peptic ulcer disease.

The trial drug was identified and authenticated by the botanist and *Gunapadam* experts.

The physico chemical analysis result showed the pH of *Panai poo chooranam* is 6.6. So it neutralizes the acidic pH of gastric juice.

The phyto chemical analysis result showed the presence of Tannins, triterpenes, steroids, glycosides, saponin and phenol.

Previous studies about tannin proved that, it had the anti ulcer property. Hence the author concluded that Panai poo chooranam has anti ulcer property.

The examination of acute toxicity carried out on mice indicated that *Panai poochooranam* has no toxicity when administered orally (up to 5g/kg). Toxicity study revealed the safety of the drug.

Clinical trial results showed that 76% of patients were having improvement in the clinical features. The drug is easily available and preparation is very simple. The trial medicine is cost effective. No adverse effects were produced during the entire course of treatment. From these results the author has concluded that the drug "*Panai poo chooranam"*( *Borassus flabellifer* Linn ) gives a new hope in the treatment of Peptic ulcer disease.

## SUMMARY

The herb *Panai poo* was collected from Soorapalli, Salem (Dt) and purified then powered and stored. This drug was subjected to various studies by the author.

*Panai poo Chooranam* was selected for this study to evaluate the Anti – ulcer activity, and to prove its efficacy and safety in peptic ulcer disease.

To collect the information about the drug, various text books, Literature were referred. From them, the author came to an idea about the drug and its efficacy on Peptic ulcer.

A brief description about botanical aspect of the herb *Panai poo* and its identifying characters and Phyto chemical data's were given.

The pharmacological analysis showed that the drug has got significant Anti ulcer activity.

In clinical study, the drug has showed marked improvement in 76% of cases.

The patients were responding well from the beginning of the treatment and no adverse effects were reported.

This present study suggests that *Panai poo Chooranam* has the remarkable medicinal value against the disease Peptic ulcer without any adverse effect.

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# INTRODUCTION

Siddha system of medicine being highly systematized medicine which has been founded by siddhars. This system of medicine is a gift to the mankind.

According to Siddha System the human body is the replica of the universe. Siddha system believes that all objects in the universe including human body are composed of five basic primordial elements, namely earth, water, fire, air and space.

The human body is a conglomeration of three humors (Vatham, Pitham, and Kapam) and seven physical components (Saaram, Senneer, Oon, Kozhuppu, Enbu, Moolai, and Sukkilam). The Food is considered to be the basic building material of human body, which gets processed into humors, tissues and wastes. The equilibrium of humors, body tissues and waste products is considered as health and its disturbance or imbalance leads to disease or pathologic state.

According to the humoral pathology, Liver diseases occur due to the derangement of Pitha humor.

Liver is the largest solid organ in the body. The liver plays astonishing vital functions in the maintenance, performance and regulating homeostasis of the body. It carries out a large number of critical functions, including manufacture of essential proteins, and metabolism of fats and carbohydrates. It also serves to eliminate harmful biochemical waste products and detoxify alcohol, certain drugs, and environmental toxins.

Maintenance of a healthy liver is essential for the overall well being of an individual.

Liver diseases are becoming one of the significant worldwide health problems, with high endemicity in developing countries.

Liver disease is a term for a collection of conditions, diseases, and infections that affect the cells, tissues, structures, or functions of the liver.

Liver is continuously exposed to lots of toxins, carcinogenic agents, infections, alcohol, and many other harmful substances. This will lead to liver diseases. Jaundice is often seen in liver disease such as hepatitis or liver cancer.

**Types of liver disease are,**

- Liver disorders due to impaired metabolic function. Generally the disorders associated with fat (liposis) and bilirubin (Jaundice) metabolisms are very commonly seen.
- Disorders associated with fat metabolism - Fatty Liver
- Classification of Jaundice
    - Hepatocellular jaundice
    - Hemolytic
    - Obstructive
- Hepatitis – may be viral, toxic or deficiency type.
- Chemical / Drug induced Hepatotoxicity:
- Cirrhosis.
- Necrosis.
- Hepatic failure.

**Jaundice is one of the important signs of liver disease.**

**Long-term effects of liver disease include:**

Cirrhosis of the liver, Liver failure, Illnesses in other parts of the body, such as kidney damage or low blood counts, Gastrointestinal bleeding, Encephalopathy, Peptic ulcers, Liver cancer.(Text book of medicine- K V Krishna doss).

According to the latest WHO data published in April 2011 Liver Disease Deaths in India reached 208,185 or 2.31% of total deaths. The age adjusted Death Rate is 23.59 per 100,000 of population ranks India #27 in the world.

Study estimated the annual incidence of jaundice cases as 244 (95% CI201-287) per 100,000 population. Almost 95% jaundice cases occurred in summer and monsoon months. People from all socio-economic strata were affected. (WHO 2011)

Jaundice cases were reported to be 1361 persons per 100,000 populations. (WHO 2011)

Treatment options for common liver diseases are challenging. The effectiveness of treatments such as interferon, colchicine, penicillamine, and corticosteroids are inconsistent. Treating hepatitis B, hepatitis C, and hepatitis D may involve the use of medications such as the antiviral medication alpha interferon. Side effects of interferon include a flu-like illness with fever, and body aches. Other medications used to treat liver disease may include ribavirin, lamivudine, steroids, and antibiotics. Antibiotics may cause stomach upset or allergic reactions. (Bowman W.G, Rand M.J., Text book of Pharmacology)

A liver transplant can cause many complications, including failure or rejection of the new liver. After a liver transplant, a person will need to take powerful anti-rejection medications for the rest of his or her life. Because these medications interfere with normal immunes system functioning, they increase the person's risk for infections and certain types of cancer.

Hence, there is an ever increasing need for safe hepatoprotective agent.

A drug having beneficial effect on the liver is known as hepatoprotective drug. Hepato protection is the ability to prevent damage to the liver.

Despite the significant popularity of several herbal medicines for liver diseases they are still unacceptable treatment modalities for liver diseases. The limiting factors include Lack of standardization of the herbal drugs, identification of active principles, clinical trials, toxicological evaluation.

Siddha medicines offer cure to varied spectrum of ailments. They are mysterious of curing and healing as well as preventing diseases.

Herbal drugs are significant source of hepatoprotective drugs. Mono and poly-herbal preparations have been used in various liver disorders.

In siddha literature *Sarabendra Vaithiya rathnaavali* the herbo mineral preparation *Arithiraadh*i *Chooranam* is indicated for Jaundice. *Arithiraadhi chooranam* contains Curcuma longa, Terminalia chebula, Terminalia bellerica, Phyllanthus emblica, Picrorhiza kurrao, Sodium chloride impure (Rock salt).

Hence the author is very much interested to conduct a detailed scientific study including toxicological profile, pre clinical and clinical trial to explore the value of siddha system of medicine.

# 2. AIM AND OBJECTIVES

## Aim

In the modern world Liver diseases are the most serious ailment. Plant drugs are known to play a vital role in the management of liver diseases. There are numerous plants and polyherbal formulation claimed to have hepatoprotective activities. The ultimate aim of this study is to evaluate the scientific validity of ***Arithiraadhi chooranam*** **(**AC) for hepatoprotective activity.

## Objectives

In this dissertation work, the "***Arithiraadhi chooranam*****"** is analyzed to assess the following aspects:

- Collecting the literature evidences related to the trail drug
- Getting the proper authentication of Raw drugs
- Preparation of the trial drug, according to the text in a sasthric manner
- Physico-chemical, Chemical Analysis for the trial drug to identify the active components.
- Toxicological studies to prove the safety of the drug.
- Pharmacological study to evaluate the hepatoprotective efficacy of the drug
- Clinical studies Evaluating the therapeutic efficacy of ***Arithiraadhi chooranam*** through open clinical trial on ***kamalai*** patients

# 3. REVIEW OF LITERATURE

The literature support for my trial drug were collected from the classical literatures of Siddha, Siddha text books, Wealth of India, Materia medica, Compendium of Medicinal Flora, and MAPA.

## 3.1.Botanical aspect

### *Manjal - Curcuma longa*

**Scientific classification**

Kingdom : Plantae

Division : Magnoliophyta

Class : Liliopsida

Order : Zingiberales

Family : Zingiberaceae

Genus : *Curcuma*

Species : *C.longa*

## 3.2 Gunapadam aspect

### Vernacular names

Eng : Turmeric

Tamil : *Arisanam, kansani, Nisi, Peedham*

San : *Haridra*

Tel : *Pasupu*

Mal : *Mannal*

Kan : *Arisina*

**Part used** : Rhizome

Taste : Acrid, bitter

*Thanmai* : *vepam*

*Pirivu* : *karpu*

**Actions**

Hepato tonic

Stimulant

Carminative

Aromatic

பொதுகுணம்

பொன்னிறமாம் மேனி புலானாற்ற மும்போகும்

மன்னு புருட வசியமாம் – பின்னியெழும்

வாந்திபித்த தோடமையம் வாதம்போந் தீபனமா

கூர்ந்தமஞ்ச லின்கிழங்குக்கு.

தலைவலிநீ ரேற்றஞ் சளையாத மேகம்

உலைவுதரு பீனசத்தி னூடே - வலிசுரப்பு

விஞ்சு கடிவிடமும் வீறுவிர ணங்களும்போம்

மஞ்சள் கிழங்குக்கு மால்.

அகத்தியர் குணவாகடம்

## Medicinal uses

- Turmeric used by externally gives golden colour to the body.
- It removes bad odour.
- Stimulates appetite.

- It cures vomiting, *vadha, pitha* disorders, headache, Leucorrhoea, running nose, 5 types of *vali*, oedema, insect bite, wounds.
- The powdered form of turmeric used as externally cures wounds.
- The burnt form of turmeric, when inhaled relives sinusitis.
- It is grounded with neem leaves, and applied externally for chicken pox.
- It is grounded with *adadhoda* leaves and cow's urine, it cures scabies, itching.
- Fresh turmeric juice cures leech bite, and wounds.
- 520-650mg of turmeric powder gives internally, it will cure stomach disorders. It also strengthens the intestine.
- **Water mixed with turmeric and consumed, it cures jaundice**.

## Phyto chemical constituents

- Rhizome has phenolic compound called curcumin, and a volatile oil with turmerone and zingiberene, cineole and other monoterpenes; starch; protein; and high amounts of vitamin A and other vitamins.
- The essential oil stimulates the gallbladder and also stimulates the liver to produce more bile and regulate its viscosity.
- Antioxidants including vitamins C and E, several carotenoids, curcumin, and related compounds called curcuminoids.
- Turmeric also has anti-inflammatory and strong liver-protecting properties.
- Cineole which stimulates the central nervous system. It is also an antiseptic and expectorant.

Wealth of India Volume- 3

***Nelli mulli - Phyllanthus emblica***

## 3.1 Botanical aspect

### Scientific classification

Kingdom : Plantae

Division : Angiosperm

Class : Eudicots

Order : Malpighiales

Family : Phyllanthaceae

Genus : Phyllanthus

Species : P.emblica

## 3.2 Gunapadam aspect

### Vernacular names

Eng - Indian gooseberry

Tam - *Nellikai*

Sans - *Amaraphalam, Amalaki*

Tel - *Nelli*

Hind - *Amla*

**Part used** : Dried fruit

**Actions**

Refrigerant

Diuretic

Laxative

Taste : *Pulipu, thuvarpu, inipu*

*Thanmai* : *Thatpam*

*Pirivu* : *Inipu*

## நெல்லிமுள்ளியின் குணம்

ஆகவன லஞ்சசிஅ சிர்க்கென்பு ருக்கிகண்ணோய்
தாக முதிரவித்தந் தாது நஷ்டம்- மேகனத்தின்
இல்லிமுள்ளி போலருகல் எண்கா மியவியங்கம்
நெல்லிமுள்ளி யாற்போ நினை.

தேரையர் குணவாகடம்

நல்லநெல்லி முள்ளியது நாக்குக் குருசிதரும்
அல்லல்விரி பித்தம் அகற்றுமதை - மெல்லத்
தலை முழுகக் கண்குளிருந் தாவுபித்த வாந்தி
இலையிழிமே கங்களும் போம் எண்.

தேரையர் குணவாகடம்

நெல்லிமுள்ளியால் உட்சூடு, எலும்புருக்கி, குருதியழல், பெரும்பாடு, வெறி நோய், நீரருகல், வாந்தி, வெள்ளை, ஆண்குறி கொப்புளம், ஆகியவை விலகும்.

**Medicinal uses**

**Nellikai legiyam and nelli mulli legiyam cures jaundice.**

***Kadugurohini - Picrorrhiza kurroa***

### 3.1 Botanical aspect

**Scientific Classification**

Kingdom : Plantae

Division : Magnoliphyta

Class : Magnoliopsida

Order : Solamaceae

Family : Scrophulariaceae

Genus : *Pirorhiza*

Species : *P. kurrao*

### 3.2 Gunapadam aspect

**Vernacular names**

Eng : Picrorhiza

Tam : *Kadugurohini*

Sans : *Katurohini*

Hind : *Katuka*

Tel : *Katki*

**Part used** : Dried rhizome

Taste : *kaipu, karpu*

*Thanmai* *: vepam*

*Pirivu* *: karpu*

**Actions**

Antiperiodic

Cathartic

Stomachic

Anthelmintic

## பொதுகுணம்

மாந்தம் சுரமையம் வாயுகரப் பானாமஞ்

சேர்ந்தமலக் கட்டு திரிதோடம் - போந்தபொட்டுப்

புண்வயிறு நோயிவைபோம் பொற்கொடியே பேதியுண்டாம்

திண்கடுகு ரோகணிக்குத் தேர்.

அகத்தியர் குணவாகடம்

மாந்தம், சுரம், ஐயம், கரப்பான், சீதகழிச்சல், வயிற்றுவலி, புண்கள், வளி நோய்கள் தீரும்.

**Phyto chemical Constituents**

Root contains iridoid glycosides, glucoside called Picrorhizin, picroside I, picrosideII, kutkoside, picroside III, veronicoside, minecoside, phenol, cucurbitacin glycosides, glucose, wax, Cathartic acid etc.

Picrorhiza prevented damage caused to the liver by intraperitoneal administration of carbon tetra chloride.

Kokate Text book of Pharmacognosy

***Kadukkai - Terminalia chebula***

## 3.1 Botanical aspect

### Scientific classification

Kingdom : Plantae

Division : Magnoliophyta

Class : Magnoliopsida

Order : Myrtales

Family : Combretaceae

Genus : *Terminalia*

Species : *T. chebula*

## 3.2 Gunapadam aspect

### Vernacular names

Eng : Chubulic myrobalan

Tam : *kadukai*

Sans : *Pathya*

Hind : *Pile hara*

### Actions

Astringent

Alterative

Laxative

### பொதுகுணம்

தாடை கழுத்தக்கி தாலு குறியிவிடப்

பீடை சிலிபதமுற் பேதிமுடம் – ஆடையெட்டாத்

தூலமிடி புண்வாத சோணிகா மாலையிரண்

டாலமிடி போம்வரிக்கா யால்.

அகத்தியர் குணவாகடம்

கன்னம், கழுத்து, நா, ஆண்குறி நோய்கள், காலடிப்புற்று, அதிதூலம், இடிப்புண், வாத சோணித வாதம், காமாலை, தாவர சங்கம விடங்கள் போகும்.

**Phytochemical Constituents**

Tannin, gallic acid, Chebulinic acid, mucilage

***Thanrikkai - Terminalia bellerica***

## 3.1 Botanical aspect

## Scientific classification

Kingdom : Plantae

Division : Magnoliophyta

Class : Magnoliopsida

Order : Myrtales

Family : Combretaceae

Genus : *Terminalia*

Species : *T. bellerica*

## 3.2 Gunapadam aspect

## Vernacular names

Eng : Belleric myrobalan

Tam : *Thanrikai*

Sans : *Vebeethaki*

Hind : *Bhairah*

## Actions

Astringent

Expectorant

Laxative

Tonic

**Part used** : Fruit

Taste : *Thuvarpu*

*Thanmai* : *Vepam*

*Pirivu* : *Inipu*

## பொதுகுணம்

சிலந்திவிடம் காமியப்புண் சீழான மேகங்
கலந்துவரும் வாதபித்தங் காலோ – டலர்ந்துடலில்
ஊன்றிக்காய் வெப்ப முதிரபித் துங்கரக்குந்
தான்றிக்காய் கையிலெடுத் தால்.
ஆணிப்பொன் மேனிக் கழகும் ஒளியுமிகும்
கோணிக்கொள் வாதபித்தக்கொள்கைபோம் – தானிக்காய்
கொண்டவர்க்கு மேகமறும் கூறா அனற்றணியும்
கண்டவர்க்கு வாதம்போம் காண்.

அகத்தியர் குணவாகடம்.

தான்றிக்காயால் சிலந்தி நஞ்சு, ஆண்குறிப்புண், வெள்ளை, குருதியழல், வளி தீ குற்றங்களால் வரும் நோய்கள் போம். உடற்கு அழகையும் ஒளியையும் கொடுத்து முக்குற்றங்களையும் தன்னிலைப்படுத்தும்.

**Phytochemical Constituents**

Gallotanic acid, resins, colouring matter, greenish yellow oil

Wealth of India Vol-7

***Indhuppu* - Sodium chloride impura**

**3.2 Modern aspect**

**Chemical Name: Sodium chloride – NaCl**

**Source**

Found in nature in extensive beds mostly associated with clay and calcium sulphate. To obtain it, holes are dug into these rocks which soon become filled up with salt water, the water is evaporated and the salt is left ready for use.

**Characters**

It is found in small white crystalline grains or transparent cubes. It is brownish white externally and white internally. It has a pure saline taste and burns with a yellow flame.

**Action**

Carminative, Stomachic, and digestive.

It promotes the appetite.

Cathartic and Emetic (in large doses)

**Uses**

- ❖ It is given in dyspepsia and other abdominal disorders.
- ❖ A powder containing *pancha lavana* 5 parts impure oxide of iron 10 grains in dyspepsia, congested liver.
- ❖ Medicated oil named *Salpa Masha Taila* is used as an application in rheumatism, contracted knee joint, stiff shoulder joint.

Indian material medica Vol - 7

## 3.1 Gunapadam aspect

### Vernacular names

Eng : Rock salt

Tam : *Indu uppu*

Sans : *Saindhava*

Hind : *Sendhalon*

Tel : *Saindhalavanam*

Mal : *Intu uppu*

Guj : *Sindhaluna*

### பொதுகுணம்

அட்டகுன்ம மந்தம் அசிர்க்கரஞ்சூர் சீதபித்தந்

துட்டவையம் நாடிப்புண் டோடங்கள் – கெட்டமலக்

கட்டுவிட விந்தையக் காமியனோய் வன்கரப்பான்

விட்டுவிட விந்துப்பை விள்.

சென்னிக்கண்ணா பற்றூர் செவிகவுள்கண் டம்பகநோய்

சந்நியா சங்காசந் தாகமிரைப் – புன்னிரத்த

மூலஞ் சிலந்திநளி மூடிகநஞ் சூதை வலி

சூலஞ் சிதையுமிந்தாற் சொல்.

குணபாடம் தாது வகுப்பு

இந்துப்பினால் எண்வித குன்மம், அலசம், அசிர்க்கரம், கபபித்தம், கபாதிக்கம், நரம்புகிரந்தி, திரிதோடம், மலபந்தம், விடம், சுக்கிலம், கப உபதம்சம், கடுவன், தலை, விழி, நா, தந்தமூலம், தாது, கன்னம், கண்டம், யோனி நோய்கள், சந்நியாசம், நேத்திர காசம், தாகம், சுவாசம், ரத்த மூலம் முதலிய பிணிகள், சிலந்தி, தேள், எலி இவற்றின் விடங்கள், வாதக் கடுப்பு, சூலை முதலியன நீங்கும்.

**Other Tamil names**

*Saindhavam, Sindhooram, chandhiranuppu, Madhikoormai, Madhiyuppu, Mindhasol*

**Actions**

Carminative, Stomachic, digestive, cathartic and emetic

**Occurrence**

In *sindhu desam* and North West part of Punjab rock salt is mined from under ground salt deposits. The weight of this salt bar is about 2 to 10 pounds. Outer surface is *kabila niram* and inner surface is white in colour. Salt in taste.

***Vaipu murai***

Sea water -200 lit

Pottasium nitrate (*vediuppu*) -175 g

Alumen (*seenakaram*)-175g,

Fullers earth (*pooneru*) -105g

by evaporation of sea water, we get salt crystals. Take this salt 3800kg and heat it. When the salt is melting, add powder of last 3 drugs. Then cool it. Now this structure of salt is look like diamond, when broken. It ruins *Borneo camphor (pachai karpooram*). It can convert calomel (*pooram*) into *muppoo*.

Rock salt is one of the earth *bootha* elements.

## Purification

- Soak in vinegar for 3 days and dry it in sun light.
- Soak in vinegar or goat urine and dry it in sun light.

## Medicinal uses

### External

Rock salt paste is applied externally for sprain. Fomentation for painful oedema.

### Internal

Rock salt is used for emetic action give it with warm water.

### Pitha kasayam

Cassia senna
Pimpinella anisum
Dried ginger
Coriander seeds
} each -6gm

break these drugs into few pieces. Add 325 ml water and boil it till reduce into 162.5 ml. Add 8.4g to 21g rock salt with this kasayam for purgative action. This is called *Pitha kasayam*. Dr. Mohedeen shareep said that it is specially used to cure indigestion

(*akni mantham*), eye diseases(nethira rogam), *kiranthi*, *thaabanum, kaba pitham*, and rat poison.

### *i. Indhuppu chooranam*

Rock salt -1 part

*Cuminum cyminum* - 1 part

*Carum copticum* - 4 Parts

*Piper longum* -8 times

Dried ginger -16 times

*Terminalia chebula* -32 times

Make these into fine powder

Dosage - 12 g

Curable diseases: Indigestion (*akni mantham*), vomiting, ascitis (*magotharam*).

ii. Cassia senna - 1 part

Dried ginger, Piper nigram, Embelia ribes, Carum copticum, Rock salt take each one as 1/4 part and make it into powder.

Sugar - 2 1/4 times

Mix the above drugs together.

Dosage: 4.2g to 8.4g

Curable diseases: Ascitis, abdominal pain, constipation (*mala bantham*).

*Indhuppu* is also an ingredient in *uppu mandooram*, *sanga thravagam* which cure anemia & ascitis.

Other Preparations containing Indu uppu curing jaundice are,

1. ***Sarabaraasa mathirai***

   Adjuvent: Mixture of *Vizhudhi saaru*, honey, ghee

   *Anuboga vaithiya navaneedham*, 7th part, Pg no- 136

2. ***Gowri Sinthaamani rasa Chendooram***

   *Anuboga vaithiya navaneedham*, 7th part, Pg no- 112

3. ***Navarasa mezhugu***

   Adjuvent: Mixture of *kumatikkai uppu*, Castor oil, honey

   *Anuboga vaithiya navaneedham*, 7th part, Pg no- 62

4. ***Kowsikar Kuzhamb* -** Adjuvent: Castor oil

   *Siddha Vaithiya thiratu*, Pg no- 209

**Other preparations of Indu uppu**

1. Vengara mathirai - Siddha vaithiya thiratu - Pg no 45
2. Pancha lavana parpam - Siddha vaithiya thiratu - Pg no 116
3. Nayuruvi uppathi kuzhambu - Siddha vaithiya thiratu - Pg no 193
4. Sanga thiravagam - Siddha vaithiya thiratu - Pg no 298

## 3.3 Siddha aspect of the disease

***Manjal noi***

**Other names of the disease**

*Pithu noi, manjal kaamalai, kaamalaa, kaamila*

**Essential features of the disease**

In this disease, the urine, eyes, tongue and the whole body will appear yellow in colour.

**Genesis of the disease**

- If one who perform the acts which aggravate *pitha dosha*,
- If one eats foods which are too much excess in quantity, the blood will be adversely affected; this causes accumulation of bilious fluid in the blood, muscles, skin, eyes and tongue producing the disease.

## மஞ்சள் நோயின் முற்குறி குணங்கள்

பருகவே உள்ளங்கா லுள்ளங் கைகள்
பகர்முகங்கண் ணுடம்புமிக வெளுப்பு காணுங்
கருகவே கால்கைக ளோய்ச்ச லாகுங்
கனமாக நடுக்கியே இளைப்புண் டாக்குஞ்
சுருகவே மலந்தானும் வறண்டு கட்டுந்
தூயமுக மஞ்சளி நிறம தாகும்
வெருகவே வீக்கமாய்க் களைப்புண் டாகும்
மிகக்காது மந்தந்தலை கனப்புண் டாகும்.

யூகி முனி

**Prodromal symptoms**

- Excessive salivation, vomiting, bitterness of tongue, dislike to food
- Indigestion if food is taken
- Dryness of body and shrinkage of skin appearing like frog's skin
- Yellow colouration of eyes, nail, face and skin of the body

In addition to the above, the following signs and symptoms may also be seen:

- Paleness of sole of foot, hand, face and eyes
- Fatigue of upper and lower limbs
- Tremors of the body, frequent attack of tiredness
- The bowel may be constipated and the stool comes out as a darkened mass
- Excessive sleep and heaviness of head and yellow colouration of the body.

**Types of disease**

*Kaamalai* disease has been classified into **thirteen types** as follows

**1. *Vali manjal noi***

- The essential features of the disease are, flatulence, hiccough, constipation, swelling of the body, edema of face and feet.
- In addition to the above, the patient may not be in a position to perform to any job due to body fatigue.
- Body pain, swelling of upper and lower limps, lean body mass, diminision of vision with swelling of eyes, anorexia and insomnia are other features of the disease.

**2. *Azhal manjal noi***

- In this disease, the skin will appear dry and patient will suffer from insomnia, will lie down on the bed lazily.
- Indigestion of food taken, pain all over the body, frequent dyspnoea, frequent diarrhea, excessive bowel sounds, loss of strength in upper and lower limbs.

**3. *Iya manjal noi***

- The disease develops due to *kapha*.
- The essential features of the disease are, yellow colouration of whole body, uncontrolled cough, sweating of face and head, difficulty in walking and dyspnoea during walking, excessive congestion of eyes, tremors of the body, heaviness of chest.
- If patients take areca nut may develop giddiness.

**4. *Vali iya manjal noi***

- The food taken may not be digested, sweating over the body, face, nose and eyes, excessive bowel sounds with flatulence, tongue will have bitter taste.
- The tongue and eyes will appear pale, dyspnoea on fast walking, swelling of nail bed and excessive cough.

**5. *Azhal iya manjal noi***

- The body will become yellow in colour. The patient will develop vomiting and headache.
- There may be also loose motion appearing white like milk.
- Excessive thirst, indigestion, giddiness, swelling of whole body, paleness of face and limbs.

**6. *Tri-dosha jaundice***

- In this disease, a few or more signs and symptoms of the three diseases mentioned above may be seen.
- The body will be severely inflamed and produce burning sensation.

- The body will appear weak; there may be also cough, excessive thirst, hiccup, burning sensation of the body and dyspepsia.

**7. *Oodhu manjal noi***

- Discharge from the eyes, sweating in face, generalized swelling of the body with pain, loss of sensation over the body.
- The body will become hot and this causes excessive activity of the *kapha*, giddiness, tiredness, numbness and generalized anasarca.

**8. *Varattumanjal noi***

- Dryness of upper and lower limbs, unsteady gait, passing blue coloured stool and red coloured urine, burning pain during micturation, pain while eating and ageusia, night blindness and darkening of body.
- The whole body will be inflamed with burning sensation. There may be also frequent occurrence of tiredness and cough.
- Excessive thirst, throbbing pain of the whole body, flatulence, sour taste of tongue, frequent occurrence of pseudo appetite, hypothermia, distention of lower abdomen and swelling of the body.

**9. *Perumanjal noi***

- Passing of yellow coloured urine, swelling of the body with loss of strength, yellow discolouration of face upper and lower limps, eyes and uvula.
- Patient may develop dyspnoea and mental depression on noticing the severity of the disease and impotence and constipation.

**10. *Azhagu manjal noi***

- In this disease, there will be weakness of upper and lower limbs. The eyes and eyebrows will become green in colour.

- Excessive thirst, burning micturition with yellow coloured urine.
- In addition, there may be abdominal distention with indigestion and the body will appear hot like that of fire.

**11. *Sengamala manjal noi***

- In this disease, due to renal insufficiency the body will be inflamed and patient will develop fatigue.
- The urine will be concentrated and appear reddish yellow in colour, the body and nail will appear pale; the body may become dry and there may be fever.
- There may be also yellow discolouration of uvula, tongue and saliva.

**12. *Kumbamanjal noi***

- In this disease, the body will become heavy and patient may develop tiredness frequently.
- The urine will appear yellow in colour; there may be sweating of the body during evening; there may be also congestion of the eyes.
- Due to illness, the body will also shrink as if a flower garland shrinks.
- Due to severity of the illness, the patient may sleep while standing similar to that of a peacock sleeps while standing.
- Patient may also passing motion frequently similar to that of dysentery.

**13. *Gunma manjal noi***

- In this disease, the mouth will appear pale and the eyes will appear greenish in colour. The urine becomes yellow in colour and will be passed like colloid.

- During digestion pain may develop in the lower abdomen along with vomiting; patient may also develop loss of smell, frequent cough dyspnoea.

- He may also lie down without any movement like a dead body or he may also roll down in the bed due to severe abdominal pain.

**Curable and incurable disease**

Of the thirteen disease mentioned above , the following seven diseases are curable,

*Azhal Kaamaalai, manjal Kaamaalai, Kaamaalai, Oothu kaamaalai, Varattu kaamaalai, Vali iya kaamaalai and vali azhal kaamaalai.*

**Not easily curable diseases are**

*Kumba kaamaalai, Gunma kaamaalai, Tri-dosha kaamaalai, vali kaamaalai, Sengamala kaamaalai and Azhaghu kaamaalai*.

**General features of the disease**

- As soon as the disease develops, the sclera of the eye will become yellow in colour. After that face, neck and body will become yellow in colour. As the disease progresses, the upper portion of the mouth, lips and urine will also become yellow in colour. Finally, the skin will appear yellowish green in colour.

- When the severity of the disease increases there may be bleeding from the nose, gums and mouth.

- The urine will appear dark yellow in colour as if haematuria has occurred. If the urine has fallen on the cloth, it will cause stain.

- Even the sweat will appear yellow in colour.

- The stool will appear pale and will be passed like a gum.

- In some patients, even the saliva, tears and teeth will also appear yellow in colour.
- In addition to the above, when the disease develops, the body will become dry and the skin will appear as if frog's skin.
- There may be also itching, the gait will be unsteady and also suffer from insomnia and all the objects will appear to him yellow in colour.

**Tri-dosha factors**

The *pitha dosha* becomes excessive in activity due to certain diets which aggravate *pitha dosha*, walking under the sun rays and awakening at night.

This *pitha dosha* in association with *kapha dosha* adversely affects the function of directional factors and impairs the potency of the blood and causes the disease .

There may be also excessive secretion of bilious fluid which will not be metabolized naturally, the excess bilious fluid in the blood produce a lot of adverse effects.

Due to association of *pitha* and *kapha*, the eyes, ears and stool will attain yellow colour.

## 3.4 Modern aspect of the disease

**Jaundice**

Jaundice is the yellow discolouration of sclera, mucous membranes, and skin caused by accumulation of bilirubin. Jaundice results from either excessive production or defective elimination of bilirubin. The level of serum bilirubin has to reach approximately 3 mg/dl before such a change is noted clinically.

There are classically three types of jaundice. These are:

**1. Obstructive (post hepatic) jaundice**

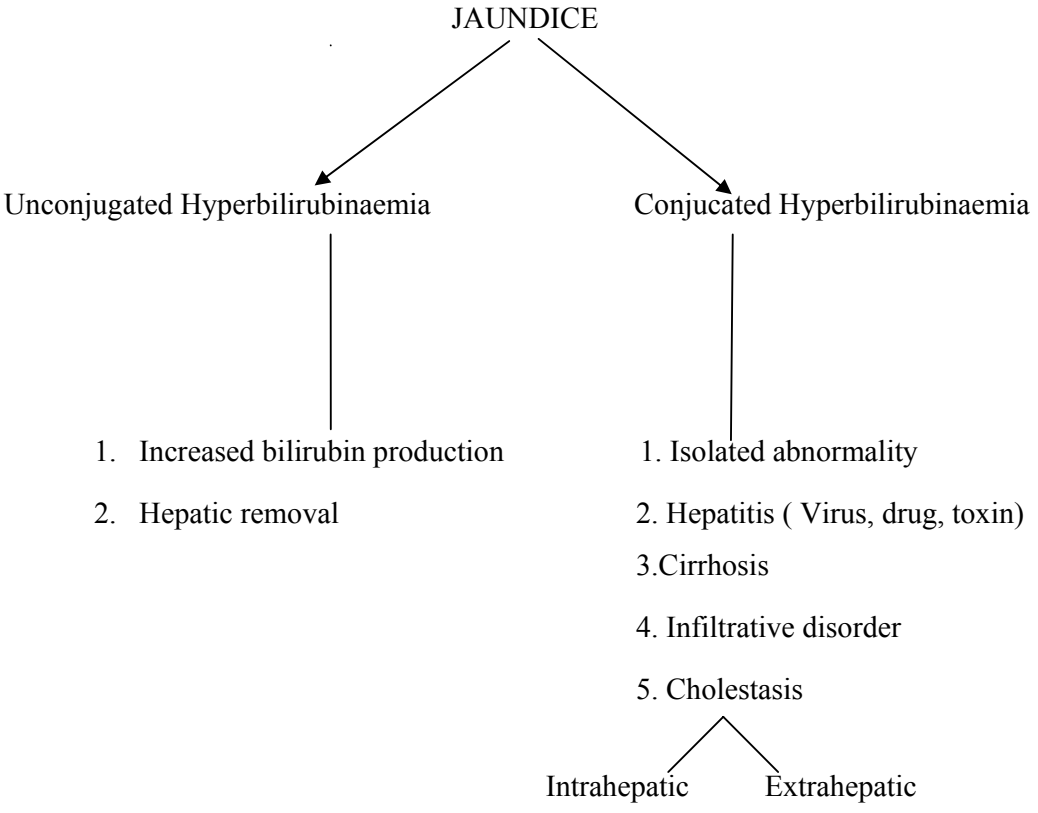
In this condition there is obstruction either in the common bile-duct, both hepatic ducts, or in the biliary ductules or canalicu inside the liver. It can therefore be classified as either intra-or extra hepatic.

**2. Liver cell (Hepato cellular) jaundice**

There is a failure of the liver cell to take up and conjugate bilirubin and to deliver it to the biliary canaliculi for excretion.

**3. Haemolytic (Pre hepatic) jaundice**

In this situation which is accompanied by haemolytic anaemia the amount of bilirubin delivered to the liver is in excess of its excretory capacity. It therefore collects in the serum in the unconjugated form.



The main causes of unconjugated hyperbilirubinaemia are,

1. Gilbert's syndrome
2. Crigler-Najjar type I
3. Crigler-Najjar type II
4. Physiological jaundice of the newborn
5. Transient familial neonatal hyperbilirubinaemia
6. Breast milk jaundice

Causes for conjucated hyperbilirubinaemia are,

1. Dubin-johnson syndrome
2. Rotor syndrome

**Pathophysiology of jaundice:**

Hyperbilirubinaemia with jaundice may result from excessive bilirubin production, reduced hepatic uptake of bilirubin, reduced hepatic conjugation of bilirubin, or reduced excretion of conjugated bilirubin.

**Excessive production of bilirubin**

Excessive production of bilirubin occurs in the haemolytic anaemias. It also may occur in patients with a marked degree of ineffective erythropoiesis and intramedullary destruction of red cell precursors, as occurs, for example, inthalassaemia and pernicious anaemia.

**Reduced uptake of bilirubin**

A defect in the transfer of bilirubin to the hepatocyte may explain the cnconjugated hyperbilirubinaemia associated with Gilbert's syndrome, severe congestive heart failure, portocaval shunts, and the action of certain drugs.

**Obstructive Jaundice**

**Clinical picture**

Patients with obstructive jaundice have usually experienced a gradual onset of the condition, as serum bilirubin levels take some days, or longer, to reach a maximum. The jaundice is of variable intensity, but with complete obstruction serum bilirubin levels may be 30 mg/dl or more.

The jaundice is usually accompanied by persistent itching, though this is not invariable, and this phenomenon is thought to be due to the deposition of irritant bile salts in the skin. The patient may complain of pain, particularly if the cause lies in the obstruction of the common bile-duct by gallstones or by a growth at the head of the pancreas.

In a similar way there may be gross weight loss in patients with neoplastic disease. Pale stools and dark urine are universal accompaniments of this form of jaundice.

The intrahepatic causes of obstructive jaundice include various drugs, one form of viral hepatitis (Obstructive hepatitis) and wide intrahepatic spread of malignant tissue as is sometimes seen in patients with carcinomatosis or Hodgkin's disease.

The physical signs of obstructive jaundice include icterus and hepatomegaly. In chronic cases the spleen may become enlarged, and in patients with neoplastic disease enlargement of supraclavicular glands on either side of the neck may be a feature.

Rectal examination may reveal secondary deposits in the pouch of Douglas, and this is an important part of the physical examination of the jaundiced patient.

It is also important in patients with chronic obstruction to look for deposits of cholesterol, particularly around the eyelids (xanthelasma) and on bony points or sites of pressure. An important physical sign is occasionally seen in patients with neoplastic obstruction of the common bile-duct below the cystic duct and this is the presence of a spherical tumour felt under the hepatic free margin, which is an

enlarged gall-bladder. This is called courvoisier's sign and is not seen in patients with obstructive jaundice due to biliary calculi.

Masses may be found in the abdomen suggesting a primary neoplasm in the stomach, colon or elsewhere, and in such patients' examination of the stools for occult blood and precise localization by radiology are important.

**Diagnosis**

Characteristically, in obstructive jaundice the liver function tests show a raised serum bilirubin of variable extent, a high percentage of conjugated bilirubin in the serum, Negative flocculation tests, raised $\alpha_2$ and $\beta$ globulins, and moderately raised transaminases, though these may be more elevated in patients with chronic obstruction due to accompanying liver-cell dysfunction.

Most important of all, there is a raised serum alkaline phosphatase usually in excess of 30KA units/dl.

The urine contains bile and in cases of complete obstruction is free of urobilinogen. The stools are pale because of lack biliary pigment and accompanying steatorrhoea.

The serum cholesterol is raised and a specific lipoprotein, LPX, can be demonstrated in the serum.

The differentiation of intra-from extra –hepatic biliary obstruction very much depends on the demonstration in the first of the patency and non-dilation of the biliary tree by ERCP or transhepatic cholangiography.

Drugs which cause obstructive jaundice include the phenothiazines. Rare examples are due to the contraceptive pill, anticoagulant drugs, butazolidine, chlorpropamide, and amitriptyline.

### Liver Cell Jaundice

#### Aetiology

The usual causes of this disease are virus hepatitis and action of certain poisons and drugs. Ccl4 which is used in fire extinguishers and dry cleaning causes severe fatty infiltration of the liver and other organs.

Other examples of drugs causing liver cell injury include the monoamine oxidase inhibitors used in the treatment of depression and the anaesthetic agent halothane.

#### Clinical picture

The onset of this type of jaundice is usually rapid, the degree of jaundice increasing to a maximum within a day or two of the onset of the disorder.

The patient often feels unwell; there is not usually persistent itching. On examination the liver may be enlarged or decreased in size and there may be splenomegaly.

Signs like, spider naevi, palmar erythema, gynaecomastia, and clubbing are important. If liver cell jaundice is severe, the patient may show evidence of a bleeding tendency and there may be evidence of hepatic encephalopathy and oedema formation, the later due to fall in the serum albumin levels.

#### Diagnosis

The liver function tests apart from showing high serum bilirubin show abnormal flocculation tests, a rise in the β and γ globulins, normal or raised alkaline phosphatase, and most important of all, considerable elevation of the transaminases.

In patients with acute liver cell disease associated with necrosis there may be a polymorphonuclear leucocytosis in the peripheral blood. The urine contains bile pigment and an excess of urobilin or urobilinogen may be demonstrated.

### Haemolytic Jaundice

The patient with haemolytic jaundice shows only mild icterus and the urine does not contain bile pigment. It does, however, contain excessive urobiligen, and may be darker than normal, as may the stools.

The liver function tests are normal unless the patient has developed biliary calculi associated with chronic haemolysis and these may then cause a picture of biliary obstruction with raised alkaline phosphatase. The patient may show splenomegaly and there may, on occasions, be hepatomegaly.

**Diagnosis**

Examination of the blood will show other features suggestive of haemolysis, such as macrocytes and reticulocytes in the peripheral film.

Examination of the bone marrow in patients with haemolysis shows evidence of erythroid hyperplasia.

Important tests to determine the nature of the haemolytic process include a history of taking of drugs, examination of the red cells for the detection of antibody coating, an L.E. cell preparation, an electrophoresis of the haemoglobin, and a blood culture.

**Table No:3.4**

**Laboratory features of jaundice**

| Laboratory Studies | Hemolytic | Obstructive | Hepatocellular |
|---|---|---|---|
| Serum bilirubin | Indirect | Direct | Biphasic |
| Urine bilirubin | Negative | Positive | Positive |
| Urine urobilinogen | Increased | Low or absent | High |
| Stools | Dark | Clay-coloured | Pale |
| Flocclation and turbidity tests | Negative | Negative ( early) | Positive |
| Serum alkaline phosphatase | Normal | Increased | Normal |
| Total serum cholesterol | Normal | Increased | Decreased |

[ Ref: Oxford Text book of Medicine, Ronald's text book of Medicine, Robbins patholoy]

# 4. MATERIALS & METHODS

## Materials

***Arithiraadhi chooranam*** has been selected from the classical *siddha* literature, "***Sarabendhira vaithiya rathnaavali".*** Ingredients of the test drug are as follows:

1. *Curcuma longa*
2. *Terminalia chebula*
3. *Terminalia bellerica*
4. *Phyllanthus emblica*
5. *Picrorhiza kurao*
6. *Sodium chloride*

## 4.1 Preparation of chooranam

### 4.1.1 Collection of drugs

*Curcuma longa*

250 gram of the raw drug was collected from the Tampcol raw drug store at Chennai. The adulteration particles were cleaned well.

*Terminalia chebula*

250 gram of the raw drug was collected from the Tampcol raw drug store at Chennai. The adulteration particles were cleaned well.

*Terminalia bellerica*

250 gram of the raw drug was collected from the Tampcol raw drug store at Chennai. The adulteration particles were cleaned well.

*Phyllanthus emblica*

250 gram of the raw drug was collected from the Tampcol raw drug store at Chennai. The adulteration particles were cleaned well.

*Picrorhiza kurao*

250 gram of the raw drug was collected from the Tampcol raw drug store at Chennai. The adulteration particles were cleaned well.

Sodium chloride

250 gram of the raw drug was collected from the Tampcol raw drug store at Chennai.

All materials were identified and confirmed by the Head of the Gunapadam department, GSMC, Chennai.

### 4.1.2 Purification of the raw materials

*Curcuma longa*

Skin was removed, and used.

*Terminalia chebula*

Seeds were removed, and rinds part only used.

*Terminalia bellerica*

Seeds were removed, and rinds part only used.

*Phyllanthus emblica*

Seeds were removed, and rinds part only used.

*Picrorhiza kurao*

Its root was soaked in neem leaf juice, kept in sunlight, and then dried.

*Indu uppu:*

Soaked in vinegar for 3 days, and dried in sunlight.

### 4.1.3 Method of Preparation of *Arithiraadhi chooranam*

After purification process, each material should be complete dried and was powdered separately by grinding method. Those powders were sieved by white cloth (*Vasthirakayam).*

**Preservation**

The purified *Chooranam* was stored in a clean, air tight glass container.

**Life span**

3 Months.

**Administration of the drug:**

| | |
|---|---|
| Form of the medicine | : Chooranam |
| Route of Administration | : Enteral |
| Dose | : 500 mg |
| Anubanam (Vehicle) | : Warm water |
| Times of Administration | : Two times a day; before food |


**Figure No: 4.1.1**

***Curcuma longa***


**Figure No: 4.1.2**

**Terminalia bellerica**


**Figure No: 4.1.3**

**Picrorhiza kurao**


**Figure No: 4.1.4**

**Terminalia chebula**


**Figure No: 4.1.5**

**Phyllanthus emblica**

**Figure No:4.1.6**

***Indhuppu***

**Figure No: 4.1.7**

***Arithiraadhi chooranam***

## 4.2 Standardization of *Arithiraadhi chooranam*

### 4.2.1 Physico-chemical Investagations

Physico-chemical studies like total ash, water soluble ash, acid insoluble ash, water and alcohol soluble extract, loss on drying at 105°C and pH as per the WHO guide lines in Central Research Institute For Siddha.

**Determination of Total Ash**

Incinerate about 2 to 3 g accurately weighed, of the ground drug in a tared platinum or silica dish at a temperature not exceeding 450º until free from carbon, cool and weigh. Calculate the percentage of ash with reference to the air-dried drug.

**Determination of Acid Insoluble Ash**

Boil the ash obtained for 5 minutes with 25 ml of dilute hydrochloric acid; collect the insoluble matter in a Gooch crucible or on an ash less filter paper, wash with hot water and ignite to constant weight. Calculate the percentage of acid-insoluble ash with reference to the air dried drug.

**Determination of Alcohol Soluble Extractive**

Macerate 5 g of the air dried drug, coarsely powdered, with 100 ml of Alcohol of the specified strength in a closed flask for twenty-four hours, shaking frequently during six hours and allowing to stand for eighteen hours. Filter rapidly, taking precautions against loss of solvent, evaporate 25 ml of the filtrate to dryness in a tarred flat bottomed shallow dish, and dry at 105º, to constant weight and weigh. Calculate the percentage of alcohol-soluble extractive with reference to the air-dried drug.

**Determination of Water Soluble Extractive**

Proceed as directed for the determination of Alcohol-soluble extractive, using chloroform water instead of ethanol.

**Determination of Moisture Content (Loss on Drying)**

Procedure set forth here determines the amount of volatile matter (i.e., water drying off from the drug). For substances appearing to contain water as the only volatile constituent, the procedure given below, is appropriately used. Place about 10 g of drug (without preliminary drying) after accurately weighing (accurately weighed to within 0.01 g) it in a tared evaporating dish. For example, for underground or unpowderdd drug, prepare about 10 g of the sample by cutting shredding so that the parts are about 3 mm in thickness.

After placing the above said amount of the drug in the tared evaporating dish dry at 105º for 5 hours, and weigh. Continue the drying and weighing at one hour interval until difference between two successive weighings corresponds to not more than 0.25 per cent. Constant weight is reached when two consecutive weighings after drying for 30 minutes and cooling for 30 minutes in a desiccator, show not more than 0.01 g difference.

**Determination of pH**

1% solution of the drug was prepared in distilled water and pH was determined using pH meter systronics digital ph meter.

**TLC estimation of *Arithiraadhi* Chooranam**

Thin-layer chromatography is a technique in which a solute undergoes distribution between two phases, stationary phase acting through adsorption and a mobile phase in the form of a liquid. The adsorbent is a relatively thin, uniform layer of dry finely powdered material applied to a glass, plastic or metal sheet or plate. Glass plates are most commonly used.

Identification can be effected by observation of spots of identical Rf value and about equal magnitude obtained, respectively, with an unknown and a reference sample chromatographed on the same plate. A visual comparison of the size and intensity of the spots usually serves for semi-quantitative estimation.

Solvent system

Toluene: Ethyl acetate (6:1.5).

TLC plate

Aluminium plate precoated with silica gel $60F_{254}$ of 0.2 mm thickness (Merck).

Developing chamber

Camag's twin trough chamber.

Visualizing reagent

Vanillin-sulphuric acid agent.

Extract Preparation

4 g of the chooranam was soaked overnight in chloroform. Boiled on a water bath for 10 mins, filtered and concentrated to 10 ml.

Procedure

The extract was applied on the TLC using capillary and developed in the solvent system. The developed TLC plate was air dried, dipped in vanillin-sulphuric acid reagent and heated in an oven at 105°C until the development of coloured spots and photograph taken.

**4.2.1.3 Fourier transform infrared spectroscopy (FTIR)**

Instrument details

| | |
|---|---|
| **Model** | **: Spectrum one: FT-IR Spectrometer** |
| **Scan Range** | **: MIR 450-4000 cm-1** |
| **Resolution** | **: 1.0 cm-1** |
| **Sample required** | **: 50 mg, solid** |

Fourier Transform Infrared Spectroscopy (FTIR) is an analytical technique used to identify mainly organic materials. FTIR analysis results in absorption spectra which provide information about the chemical bonds and molecular structure of a material.

Infrared spectrum is useful in identifying the functional groups like -OH, -CN, -CO, -CH, -NH2, etc.

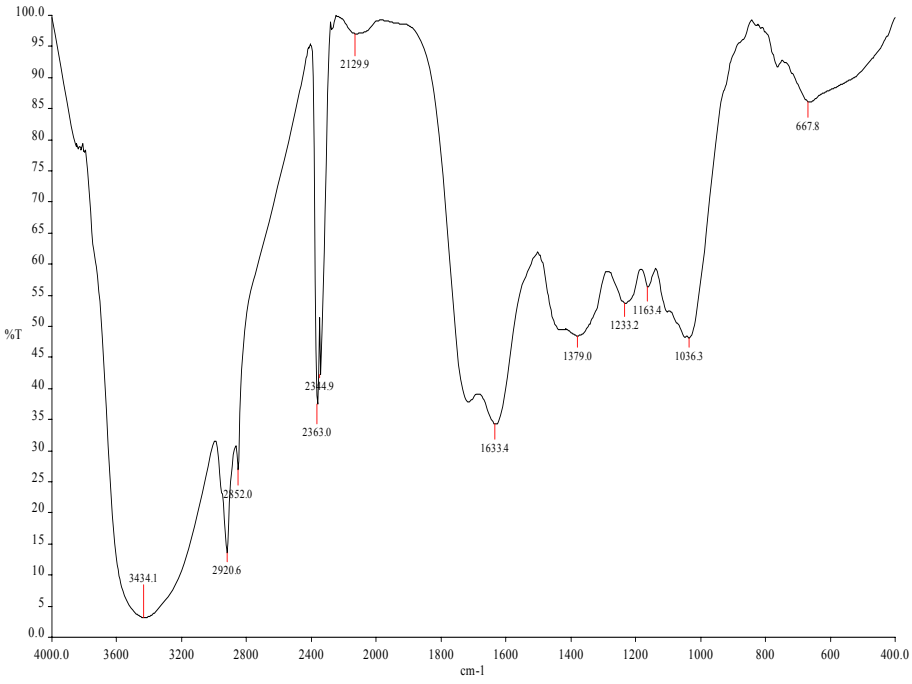



**Figure No: 4.4.1.3 Showing FTIR results.**

The drug sample was analyzed by the FTIR to identify the chemical bonds and molecular structure of a material.

### 4.2.1.4. Scanning electron microscope (SEM)

The Scanning Electron Microscope (SEM) is a microscope that was electrons rather than light to form an image. There are many advantages to using the SEM instead of a light microscope.

Resolution : 1.2 nm gold particle separation on a carbon substrate

Magnification : From a min of 12 x to greater than 1, 00,000 X

The SEM has a large depth of field, which allows a large amount of the sample to be in focus at one time. The SEM also produces images of high resolution, which means that closely spaced features can be examined at a high magnification. Preparation of the samples is relatively easy since most SEM one require the sample to be conductive.

The combination of higher magnification, larger depth of focus, greater resolution, and easy of sample observation marks the SEM one of the most heavily used instruments in research areas today.

### 4.2.2 Phyto chemical analysis of test drug

**Table No: 4.2.2**

| Sl.No | Experiment | Observation | Inference |
|---|---|---|---|
| 1. | **Test for Alkaloids:** Alkaloids are identified by precipitate method<br>**Dragendroff's reagent:**<br>An aqueous plant extract of 2 ml is mixed with 2 ml of Dragendroff's reagent. | Absence of reddish brown precipitate | Absence of alkaloids |
| 2. | **Test for Triterpenoids (Noller's Test)**<br>To few mg of extract, add tin and thionyl chloride and heat in water bath. | Presence of purple colour | Presence of Triterpenes |
| 3. | **Test for Tannins:**A plant sample dried powder 0.5 gm is boiled in 20 ml of water and filtered. The filterate 2 ml is taken and 3-5 drops of $Fecl_2$ (0.1%) is slowly added to it. | Forms a brownish-green or bluish- black colour. | Presence of Tannins |

| Sl.No | Experiment | Observation | Inference |
|---|---|---|---|
| 4. | **Test for Flavonoids:** An aqueous filterate of plant sample is mixed with 5 ml of dilute Ammonium solution and few drops of concentrated $H_2SO_4$ is slowly added through the sides of the test tube. | Yellow colour formed | Presence of flavonoids |
| 7. | **Test for Phenolic compounds:** About 2 ml of aqueous plant extract is mixed with 2 ml of $Fecl_3$ solution. | Presence of deep bluish green colour | Presence of phenolic compounds |

### 4.2.3 Chemical analysis

**Proximate Chemical Analysis of a Drug**

**Methodology For Chemcial Analysis**

**Preparation of Extract**

Add 5 gm of the sample to 50ml of distilled water. Boil the solution for 20 minutes, cool and then filter. The Extract was used for the following tests.

**Table No: 4.2.3**

| S.No | Experiment | Observation | Inference |
|---|---|---|---|
| 1. | **Test for reducing Sugar :** To 5ml of Benedicts qualitative reagent, add 10 drops of extract, then boil for two minutes | Green colour PPT | Presence of Reducing Sugar |
| 2. | **Test for Starch :** To 5 ml of extract add 2ml of acetic acid and then add few drops of N/50 Iodine Solution. | Blue Colour | Presence of Starch |
| 3. | **Test for Proteins :** To 2 ml of extract, add 2ml of 5% Sodium Hydroxide mix and add 2 drops of Copper Sulphate Solution. | Violet Colour | Presence of Proteins |

| S.No | Experiment | Observation | Inference |
|---|---|---|---|
| 4. | **Test for amino Acid :** Place 2 drops of extract on a filter paper and allow to dry well. Then spray 1% ninhydrin over the same and allow to dry. | Violet Colour | Presence of Amino Acid |
| 5. | **Test for Albumin :** To 2 ml of extract, add 2ml of Esboch's reagent. | Yellow PPT | Presence of Albumin |
| 6. | **Test for Phosphate :** To 2ml of extract, add 2ml of ammonium Molybdate solution and 2ml of concentrated Nitric Acid. | Absence of yellow PPT | Absence of Phosphate |
| 7. | **Test for Sulphate :** To 2 ml of extract add 2ml of 4% ammonium oxalate solution**.** | White PPT | Presence of Sulphate |
| 8. | **Test for Chloride :** Add 2ml of extract to dilute nitric acid till the effervescence ceases. Then add 2 ml of Silver Nitrate Solution. | Cloudy White PPT | Presence of Chloride |
| 9. | Test for Iron : To 2ml of extract, add 2ml of ammonium thio cynate solution and add 2ml of concentrated Nitric Acid. | Red Colour | Presence of Iron |
| 10. | **Test for Calcium** : To 2 ml of extract, add 2 ml of 4% ammonium Oxalate Solution. | Absence of white PPT | Absence of Calcium |
| 11. | **Test for Sodium** : Make a paste with 2 pinches of the sample with Hcl and Introduce it into the blue flame. | Absence of yellow Flame | Absence of Sodium |
| 12. | **Test for Potassium** : Add a pinch of the sample to 2 ml of Sodium Nitrate Solution. Then add 2ml of Cobal Nitrate in 20% acetic acid. | Absence of yellow PPT | Absence of Potassium |

| S.No | Experiment | Observation | Inference |
|---|---|---|---|
| 13. | **Test for Zinc** :<br>To 2ml of extract, add few drops of Sodium Hydroxide. | Absence of white PPT | Absence of Zinc |
| 14. | **Test for Magnesium** :<br>To 2ml of extract, add few drops of Sodium Hydroxide Solution | Absence of white PPT | Absence of Magnesium |
| 15. | **Test for Alkaloids :**<br>a. To 2ml of extract, add 2ml of Potassium Iodide Solution<br>b. To 2ml of extract add 2ml of Picric Acid.<br>**c.** To 2 ml of extract add 2ml of Phosphotunqstic Acid**.** | Absence of red Colour<br>Absence of yellow Colour<br>Absence of white PPT | Absence of Alkaloids<br>Absence of Alkaloids<br>Absence of Alkaloids |
| 16. | **Test for Tannic Acid :**<br>To 2ml of extract add 2 ml of Ferric Chloride Solution | Black PPT | Presence of Tannic Acid |

## 4.3 Toxicological studies

### Animals

Mice of either sex weighing 25-30g and rats weighing 210-240g were obtained from the animal house of Vels University. The animals were used with the approval of the Institute animal ethics committee and obtained from Vels University, Chennai. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-28$^0$C temperature, standard light cycle (12 h light, 12 h dark) and water ad libitum. Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation. The rats were randomly assigned to control and different treatment groups, six animals per group.

### Acute toxicity study-OECD 425 guidelines

Acute oral toxicity test for the *Arithirathi Chooranam* was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure

that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice. The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe any death or changes in general behaviour and other physiological activities. Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal.

**Acute toxicity study**

Acute oral toxicity test for the Arithirathi Chooranam was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice.

Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal. The time interval was adjusted as appropriately in case of inconclusive response. The test is simpler to implement when a single time interval is used for making sequential dosing decisions. Special attention was given during the first 4 hours and daily thereafter, for a total of 14 days, except where they need to be removed from the study and humanely killed for animal welfare reasons or are found dead.

All observations are systematically recorded and Observations include changes in skin and fur, eyes and mucous membranes, and also respiratory, circulatory, autonomic and central nervous systems, and somatomotor activity and behaviour pattern. Attention was directed to observations of tremors, convulsions, salivation, diarrhea, lethargy, sleep and coma. The principles and criteria summarized in the Humane Endpoints Guidance Document taken into consideration.

Animals found in a moribund condition and animals showing severe pain or enduring signs of severe distress was humanely killed. When animals are killed for humane reasons or found dead, the time of death was recorded. Mortality was not noticed up to 5000 mg/kg. One-tenth and one twentieth of this dose was selected as the therapeutic dose for the hepatoprotective evaluation.

***Observation of toxicity signs:*** General behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded.

**Sub-acute toxicity**

In a 28-days sub acute toxicity study, twenty four either sex (3+3) rats were divided into four groups of 6 rats each. Group I that served as normal control was administered with distilled water (p.o.) while groups II, III and IV were administered daily with the Arithirathi Chooranam (p.o.) for 28 days at a dose of 100, 250 and 500mg/kg respectively. The animals were then observed daily for gross behavioural changes and any other signs of subacute toxicity. The weight of each rat was recorded on day 0 and weekly throughout the course of the study, food and water consumption per rat was calculated. At the end of the 28 days they were fasted overnight, each animal was anaesthetized with diethylether, following which they were then dissected and blood samples were obtained by cardiac puncture into heparinised tubes. The blood sample collected from each rat was centrifuged with 3000 X g at $4^{o}$C for 10 min to separate the serum and used for the biochemical assays.

### Hematological and blood biochemical analyses

At the end of the study, all animals were kept fasted for 16-18 h and then anesthetized with anesthetic ether on the 28th day. Blood samples for hematological and blood chemical analyses were taken from retro orbital vein. Heparinized blood samples were taken for determining complete blood count (white blood cell count, differential white blood cell count, platelet count, red blood cell count, hematocrit, and hemoglobin) by semiautomated hematology analyzer. The serum from non-heparinized blood was carefully collected for blood chemistry and enzyme analysis glucose, creatinine, total protein, albumin, total and direct bilirubins, serum glutamate-oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP) were automatically determined using autoanalyzer.

### Necropsy

All rats were sacrificed after the blood collection. The positions, shapes, sizes and colors of internal organs were evaluated. The Spleen, Testes, Pancrea, Lung, Liver, Brain, Heart, Stomach, Intestine, Bone, Ovary, and Kidney tissues were excised from all rats to visually detect gross lesions, and weighed to determine relative organs' weights and preserved in 10% neutral formalin for histopathological assessment. The tissues were embedded in paraffin, and then sectioned, stained with haematoxylin and eosin and were examined microscopically.

### Statistical analysis

Values were represented as mean ± SEM. Data were analysed using one-way analysis of variance (ANOVA) and group means were compared using the Tukey-Kramer Multiple Comparison test using GraphPad Instat-V3 software. P values $< 0.05$ were considered significant.

## 4.4 Pharmacological activity

### Hepatoprotective activity of *Arithiraadhi chooranam*

The disorders associated with the liver are numerous and no effective treatment for these numerous disorders. In spite of tremendous advances in modern medicine, there are no effective drugs available that stimulate liver function, offer protection to the liver from damage or help to regenerate hepatic cells. Liver plays a major role in detoxification and excretion of many endogenous and exogenous compounds, any injury or impairment of its function may lead to several implications on one's health. Management of liver diseases is still a challenge to modern medicine. Conventional drugs used in the treatment of liver diseases are often inadequate. It is therefore necessary to search for alternative drugs for the treatment of liver diseases to replace the currently used drugs of doubtful efficacy and safety.

Liver is the key organ regulating homeostasis in the body. It is involved with almost all the biochemical pathways related to growth, fight against disease, nutrient supply, energy production and reproduction. Because of its unique metabolism and relationship to the gastrointestinal tract, the liver is an important target for toxicity produced by drugs, xenobiotics and oxidative stress. In view of severely undesirable side effects of synthetic agents and the absence of reliable liver-protective drugs in modern medicine, Siddha system of Indian medicine recommended for the treatment of liver disorders.

The liver regulates many important metabolic functions. Hepatic injury is associated with distortion of these metabolic functions. Additionally, it is the key organ of metabolism and excretion, thus it is continuously and variedly exposed to xenobiotics because of its strategic placement in the body .The toxins absorbed from the intestinal tract go first to the liver resulting in a variety of liver ailments. Thus liver ailments remain one of the serious heath problems. Modern medicines have little to offer for alleviation of hepatic diseases and it is chiefly the plant based preparations which are employed for their treatment of liver disorders. But there are not much drugs available for the treatment of liver disorders. Hence the present study was

undertaken to investigate the hepatoprotective activity of Arithirathi Chooranam against CCL4 induced liver damage in rats.

## Materials and methods

### *Drugs and chemicals*

Analytical Laboratory grade chemicals, solvents were used for the studies, which were procured from S.D. fine and span diagnostic Ltd.

### Animals

Swiss albino mice (25-30g) and male Wistar rats (150-200 g) were procured from an in-house animal facility of School of Pharmaceutical Sciences, Vels University, Chennai. The animals were housed under standard conditions of temperature (22 ± 3°C) and relative humidity (30-70%) with a 12:12 light: dark cycle. The animals were fed with standard pellet diet (Sai meera Feeds Ltd. Bangalore.) and water ad libitum. The Institutional Animal Ethics Committee (IAEC)Approved the protocol (Approval number) XIII/ VELS/ PCOL/ 12/2000/ CPCSEA/ IAEC/ 08.08.2012).

### Hepatoprotective activity

Four groups of animals containing six each were used for the study. The animals from Group I served as the normal control and received the vehicle 2% CMC at a dose of 2ml kg/d of p.o. Group II served as the control received $CCl_4$ i.p. (3ml/kg) only. Groups III & IV received the $CCl_4$ i.p. + Arithirathi Chooranam 250 and 500mg/kg orally. The standard drug silymarin was administered to Group V animals in the dose 100 mg/kg/day p.o. The treatment was continued for all the 14days. The silymarin and the Arithirathi Chooranam were administered concomitantly to the respective groups of animals.

All the animals were killed on day 14 under light ether anesthesia. The blood sample were collected separately by retro orbital bleeding into sterilized dry centrifuge tube and allowed to coagulate for 30 min at 37°C. The clear serum was separated at 2500G for 10 min and biochemical investigations were carried out assess

liver function viz., total bilirubin, total protein, serum transaminases and serum alkaline phosphates. The activities of serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and total proteins (TP) were analyzed using commercial kits, to assess the acute hepatic damage caused by CCl4.

***Histopathological studies***

After draining the blood liver samples were excised, and immediately fixed in 10% formalin, dehydrated in gradual ethanol (50-100%), cleared in xylene and embedded in paraffin. Sections (4-5 μm) were prepared and then stained with hematoxylin and eosin dye for photomicroscopic observations. Initially, the materials were fixed in 10% buffered neutral formalin for 48h and then bovine solution for 6 h. Paraffin sections were taken at 5mm thickness. The sections were examined microscopically for histopathological changes.

***Statistical analysis***

The data was represented as mean ± S.E.M. Results were analyzed statistically by one-way ANOVA followed by Dunnet's multiple comparison test using Prism software (Version 4). The minimum level of significance was set at $P < 0.05$.

**4.5 Clinical assessment**

The pre clinical study of this drug has been showed the marked hepatoprotective efficacy. The clinical study was conducted to establish the efficacy and safety of the *Arithiraadhi chooranam* for *Kamalai.*

**Objectives**

- To evaluate the hepatoprotective effect of *Arithiraadhi chooranam*.
- To explore the efficacy and safety of *Arithiraadhi chooranam* in patients with liver disorder.

**Design of the study**

Open clinical trial Phase II B

**Study centre**

Arignar Anna Government Hospital of Indian Medicine and Homeopathy, Arumbakkam, Chennai – 106.

**Study participants**

Both men and women and members of all races and ethnic groups are eligible for this trial. Treatment was administered on an inpatient/outpatient basis. The patients were selected from the In-patient and Out-patient department of Arignar Anna Government Hospital of Indian Medicine and Homeopathy, Chennai – 106.

**Number of subjects**

Number of participants will be 50.

**Selection**

50 patients from both sexes of various age groups were selected for clinical trial. Among 50 patients 40 patients were treated as out-patients, 10 patients were treated as in – patients. The selection was based on the including and excluding criteria. They were clinically diagnosed on the basis of *siddha* principles with modern laboratory findings.

**Registration process**

To register a patient, the following documents have been produced.

- Copy of required laboratory tests
- Signed patient consent form

Then I verified the eligibility and assigned a patient study number, drug dose and registered the patient on the study.

**Criteria for inclusion**

Patients with liver disease are eligible for entry to the trial if the following criteria are satisfied.

- ✓ Co operative patients
- ✓ The previous drug regimen if any have been with held for 24 hours before the clinical trial.

**Criteria for exclusion**

- ✓ AIDS
- ✓ Malignancy
- ✓ Pregnant and lactating women
- ✓ TB
- ✓ Renal diseases
- ✓ Cardio vascular disorder
- ✓ Age below 10 year
- ✓ Syphilis
- ✓ The patient requires systemic steroid for the control of symptoms

**Withdrawal criteria**

Patients were removed from study when any of the criteria listed below applies. In the absence of treatment delays due to adverse events, treatment may continue for 2 cycles or until one of the following criteria applies:

- ✓ Disease progression,
- ✓ Deterioration of vital signs with cardiac, respiratory, hepatic, renal and CNS changes.
- ✓ Intercurrent illness that prevents further administration of treatment,

- ✓ Unacceptable adverse event(s),
- ✓ Patient decides to withdraw from the study, or
- ✓ General or specific changes in the patient's condition render the patient unacceptable for further treatment in the judgment of the investigator.

**Routine examination and assessment**

The full details of history and physical examination of the patients were recorded as per the proforma .The clinical assessment was done initially at the end of 7 days, 14 days and 21 days during treatment and at the end of the 21 days follow up was done. The laboratory investigation and the physiological parameters were recorded initially at the end of the treatment and at the end of follow up as per the proforma.

**Dosage**

The trial drug *Arithiraadhi chooranam* was given in the dose of 500mg.

**Administration of the drug**

| | |
|---|---|
| Form of the medicine | : *Chooranam* |
| Route of Administration | : Enteral |
| Dose | : 500mg |
| *Anubanam* (Vehicle) | : Warm water |
| Times of Administration | : Two times a day; after food |
| Duration | : 7 weeks |

**Diet restriction and medical advice**

- ✓ The patients were instructed to follow fat free, salt free easily digestible foods.
- ✓ They were advised to take tender coconut, sugar cane juice and vegetables like radish, juice of plantain stem.

- ✓ The patient was advised to take rest. But prolonged immobilization should be avoided.
- ✓ The clinical improvement was observed and recorded in the proforma of case sheet.

**Trial conduct**

The study was conducted in compliance with the protocol approved by the Institutional Review Board, and according to Good Clinical Practice standards. No deviation from the protocol was implemented without the prior review and approval of the IRB except.

**Classification of results**

1. **Good Response**

   a. Relief of Symptoms above 75%

   b. Laboratory parameter findings towards normalcy.

2. **Fair Response**

   a. 50% to 75% relief in symptoms.

   b. Significant improvement in laboratory parameter.

3. **Poor Response**

   25% to 49% relief in symptoms and minimal improvement in laboratory parameters.

4. **No Response**

   No relief in symptoms and no significant improvement in laboratory parameters.

**Follow up**

Assessment will take for every three days before treatment and after treatment. During this period clinical assessment and laboratory investigation was carried out.

**Statistical analysis**

The data will be tabulated and analyzed by students 't' test.

**Ethical review**

The protocol and any amendments were submitted to Govt siddha medical college, Chennai – 106 (IEC) and got formal approval to conduct the study. The decision of the IEC concerning the conduct of the study was made in writing to the investigator. All subjects for this study was provided a consent form describing this study and provided sufficient information for subjects to make an informed decision about their participation in this study. This consent form was submitted with the protocol for review and approval by the IEC. The formal consent of a subject, using the IEC-approved consent form, was obtained before that subject was submitted to any study procedure. This consent form was signed by the subject or legally acceptable surrogate, and the investigator-designated research professional obtaining the consent.

**Table No: 4.5.1**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, OUT – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| Sl No | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 1. | 5735 | Chandran 50/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera. | 17.7.12 To 20.8.12 | BT | 9800 | 61 | 35 | 4 | 4 | 8 | 14 | 100 | 20 | 173 | NIL | NIL | PCS | Good |
| | | | | | AT | 9800 | 62 | 35 | 3 | 4 | 8 | 14 | 98 | 18 | 168 | NIL | NIL | NIL | |
| 2. | 2355 | Vishwanathan 36/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera. | 18.7.12 To 15.8.12 | BT | 9900 | 60 | 36 | 4 | 3 | 6 | 15 | 94 | 22 | 158 | NIL | NIL | FPC | Good |
| | | | | | AT | 9900 | 60 | 37 | 3 | 3 | 6 | 15 | 96 | 20 | 155 | NIL | NIL | NIL | |
| 3. | 3875 | Sangeetha 40/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera. | 21.7.12 To 25.8.12 | BT | 9000 | 55 | 39 | 6 | 26 | 40 | 10.5 | 112 | 19 | 165 | NIL | NIL | FPC | Poor |
| | | | | | AT | 9000 | 56 | 39 | 5 | 23 | 38 | 11 | 110 | 29 | 160 | NIL | NIL | NIL | |
| 4. | 9768 | Meganathan 39/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera. | 25.7.12 To 30.8.12 | BT | 10100 | 63 | 34 | 3 | 8 | 12 | 13 | 104 | 19 | 175 | NIL | NIL | FPC | Satisfactory |
| | | | | | AT | 10100 | 63 | 33 | 4 | 8 | 12 | 13 | 100 | 18 | 165 | NIL | NIL | NIL | |
| 5. | 3674 | Janani 37/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera. | 28.7.12 To 28.8.12 | BT | 9100 | 54 | 39 | 7 | 15 | 30 | 10 | 88 | 21 | 158 | NIL | NIL | NIL | Good |
| | | | | | AT | 9100 | 55 | 40 | 5 | 15 | 30 | 10 | 86 | 19 | 144 | NIL | NIL | NIL | |

**Table No: 4.5.2**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, OUT – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| Sl No | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 6. | 5174 | Ramakrishnan 36/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera. | 30.7.12 To 5.9.12 | BT | 10200 | 62 | 34 | 4 | 6 | 10 | 14 | 82 | 21 | 164 | NIL | NIL | PCS | Good |
| | | | | | AT | 10200 | 62 | 36 | 2 | 6 | 10 | 14 | 82 | 19 | 160 | NIL | NIL | NIL | |
| 7. | 9531 | Visalatchi 53/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera. | 31.7.12 To 3.9.12 | BT | 8800 | 56 | 38 | 6 | 15 | 25 | 12 | 88 | 19 | 190 | NIL | NIL | NIL | Good |
| | | | | | AT | 8800 | 56 | 37 | 5 | 14 | 24 | 12.5 | 86 | 20 | 185 | NIL | NIL | NIL | |
| 8. | 7105 | Vijaya 41/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 2.8.12 To 30.8.12 | BT | 9000 | 53 | 40 | 7 | 18 | 28 | 11 | 100 | 20 | 150 | NIL | NIL | FPC | Good |
| | | | | | AT | 9100 | 57 | 38 | 5 | 16 | 26 | 12 | 98 | 20 | 148 | NIL | NIL | NIL | |
| 9. | 7219 | Siraj 37/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 4.8.12 To 10.9.12 | BT | 9500 | 58 | 37 | 5 | 20 | 35 | 10 | 90 | 19 | 155 | NIL | NIL | NIL | Satisfactory |
| | | | | | AT | 9500 | 58 | 38 | 4 | 18 | 32 | 11 | 89 | 19 | 160 | NIL | NIL | NIL | |
| 10. | 8593 | Devi ammal 39/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 5.8.12 To 9.9.12 | BT | 8900 | 55 | 40 | 5 | 8 | 14 | 13 | 95 | 20 | 161 | NIL | NIL | FPC | Good |
| | | | | | AT | 8900 | 56 | 39 | 5 | 8 | 14 | 13 | 94 | 18 | 157 | NIL | NIL | NIL | |

**Table No: 4.5.3**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, OUT – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| Sl No | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 11. | 6897 | Kamaraj 41/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 7.8.12 To 14.9.12 | BT | 9900 | 60 | 35 | 5 | 4 | 8 | 14 | 98 | 21 | 168 | NIL | NIL | PCS | Good |
| | | | | | AT | 9900 | 60 | 36 | 4 | 4 | 8 | 14 | 97 | 20 | 164 | NIL | NIL | NIL | |
| 12. | 2309 | Jayaprasath 37/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 10.8.12 To 18.9.12 | BT | 10000 | 63 | 33 | 4 | 6 | 12 | 13.5 | 118 | 21 | 158 | NIL | NIL | NIL | Good |
| | | | | | AT | 10000 | 63 | 35 | 2 | 5 | 10 | 14 | 110 | 19 | 160 | NIL | NIL | NIL | |
| 13. | 8909 | Arokiya raj 39/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 11.8.12 To 19.9.12 | BT | 10100 | 59 | 36 | 5 | 8 | 14 | 13 | 98 | 18 | 172 | NIL | NIL | FPC | Moderate |
| | | | | | AT | 10100 | 58 | 37 | 5 | 8 | 14 | 13 | 100 | 20 | 170 | NIL | NIL | NIL | |
| 14. | 214 | Jeevanandham 34/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 13.8.12 To 14.9.12 | BT | 9500 | 62 | 32 | 6 | 10 | 16 | 13 | 89 | 22 | 159 | NIL | NIL | NIL | Good |
| | | | | | AT | 9700 | 63 | 32 | 5 | 10 | 16 | 13 | 88 | 20 | 165 | NIL | NIL | NIL | |
| 15. | 136 | Puhpa 36/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 16.8.12 To 20.9.12 | BT | 8900 | 54 | 40 | 6 | 15 | 30 | 10 | 95 | 16 | 162 | NIL | NIL | FPC | Good |
| | | | | | AT | 8900 | 53 | 42 | 5 | 13 | 26 | 11 | 92 | 19 | 160 | NIL | NIL | NIL | |

## Table No: 4.5.4

## CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, OUT – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI

| Sl No | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 16. | 391 | Valarmathi 42/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 20.8.12 To 21.9.12 | BT | 9200 | 57 | 37 | 6 | 20 | 43 | 12 | 85 | 19 | 160 | NIL | NIL | PCS | Satisfactory |
| | | | | | AT | 9200 | 58 | 37 | 5 | 19 | 40 | 12.5 | 85 | 20 | 159 | NIL | NIL | FPC | |
| 17. | 1469 | Prithiviraj 39/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 22.8.12 To 26.9.12 | BT | 10100 | 62 | 34 | 4 | 6 | 12 | 14 | 95 | 20 | 158 | NIL | NIL | NIL | Good |
| | | | | | AT | 10100 | 62 | 33 | 5 | 6 | 10 | 14 | 90 | 21 | 160 | NIL | NIL | NIL | |
| 18. | 1503 | Krishnaveni 34/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 24.8.12 To 30.8.12 | BT | 9500 | 55 | 40 | 5 | 15 | 25 | 11 | 86 | 20 | 159 | NIL | NIL | FPC | Good |
| | | | | | AT | 9600 | 58 | 39 | 3 | 15 | 25 | 11 | 85 | 19 | 160 | NIL | NIL | NIL | |
| 19. | 205 | Rejina mary 26/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 26.8.12 To 29.9.12 | BT | 8400 | 55 | 38 | 7 | 20 | 35 | 12 | 96 | 18 | 155 | NIL | NIL | NIL | Poor |
| | | | | | AT | 8400 | 58 | 37 | 5 | 20 | 35 | 12 | 94 | 18 | 158 | NIL | NIL | NIL | |
| 20. | 7542 | Aarumugam 51/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 29.8.12 To 5.10.12 | BT | 9500 | 62 | 33 | 5 | 10 | 16 | 13 | 108 | 21 | 165 | NIL | NIL | PCS | Moderate |
| | | | | | AT | 9500 | 61 | 35 | 4 | 10 | 15 | 13.5 | 104 | 20 | 160 | NIL | NIL | FPC | |

**Table No: 4.5.5**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, OUT – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 21. | 3841 | Kalaiselvi 36/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 8.9.12 To 15.10.12 | BT | 9300 | 54 | 39 | 7 | 18 | 30 | 12 | 99 | 21 | 162 | NIL | NIL | PCS | Good |
| | | | | | AT | 9300 | 54 | 40 | 6 | 16 | 26 | 12.5 | 98 | 19 | 158 | NIL | NIL | FPC | |
| 22. | 8444 | Jothimani 44/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 15.9.12 To 25.10.12 | BT | 10200 | 63 | 34 | 3 | 6 | 8 | 14 | 112 | 21 | 153 | NIL | NIL | PCS | Good |
| | | | | | AT | 10200 | 59 | 36 | 5 | 6 | 8 | 14 | 116 | 20 | 155 | NIL | NIL | NIL | |
| 23. | 5870 | Siva Arvinth 40/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 26.9.12 To 26.10.12 | BT | 10100 | 63 | 34 | 3 | 3 | 6 | 15 | 105 | 21 | 162 | NIL | NIL | FPC | Moderate |
| | | | | | AT | 10100 | 63 | 34 | 3 | 3 | 6 | 15 | 102 | 18 | 164 | NIL | NIL | NIL | |
| 24. | 5086 | Kanagavalli 50/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 16.10.12 To 15.11.12 | BT | 10000 | 64 | 29 | 5 | 15 | 40 | 10.8 | 101 | 20 | 199 | NIL | NIL | PCS | Good |
| | | | | | AT | 10100 | 63 | 33 | 4 | 15 | 40 | 11 | 99 | 19 | 188 | NIL | NIL | FPC | |
| 25. | 8010 | Meenakshi 30/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 18.10.12 To 30.12.12 | BT | 9200 | 58 | 36 | 6 | 35 | 60 | 10.5 | 82 | 18 | 179 | NIL | NIL | NIL | Good |
| | | | | | AT | 9300 | 61 | 33 | 6 | 30 | 55 | 11 | 80 | 21 | 181 | NIL | NIL | NIL | |

**Table No: 4.5.6**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, OUT – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 26. | 225 | Munusamy 50/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 30.10.12 To 18.12.12 | BT | 9200 | 61 | 36 | 3 | 4 | 10 | 15 | 108 | 21 | 163 | NIL | NIL | FPC | Good |
| | | | | | AT | 9300 | 62 | 34 | 4 | 4 | 10 | 15 | 104 | 20 | 161 | NIL | NIL | NIL | |
| 27. | 318 | Mallika 38/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 31.10.12 To 04.12.12 | BT | 9500 | 58 | 38 | 4 | 18 | 28 | 11 | 85 | 19 | 163 | NIL | NIL | PCS | Good |
| | | | | | AT | 9300 | 59 | 38 | 3 | 18 | 28 | 11 | 84 | 20 | 161 | NIL | NIL | NIL | |
| 28. | 947 | Naveen kumar 35/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 3.11.12 To 6.12.12 | BT | 1000 | 64 | 32 | 4 | 8 | 14 | 14 | 104 | 20 | 175 | NIL | NIL | NIL | Poor |
| | | | | | AT | 9800 | 65 | 31 | 4 | 8 | 14 | 14 | 102 | 18 | 169 | NIL | NIL | NIL | |
| 29. | 948 | Murugan 38/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 3.11.2012 To 10.12.12 | BT | 9900 | 62 | 35 | 3 | 10 | 15 | 14 | 105 | 20 | 167 | NIL | NIL | PCS | Good |
| | | | | | AT | 9900 | 61 | 35 | 4 | 10 | 15 | 14 | 110 | 21 | 168 | NIL | NIL | FPC | |
| 30. | 1379 | Vasanthi 30/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 05.11.12 To 6.12.12 | BT | 8800 | 56 | 39 | 5 | 14 | 22 | 12 | 97 | 18 | 159 | NIL | NIL | FPC | Satisfactory |
| | | | | | AT | 8800 | 57 | 37 | 6 | 14 | 22 | 12 | 98 | 19 | 160 | NIL | NIL | NIL | |

**Table No: 4.5.7**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, OUT – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb Gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 31. | 1973 | Daisy 30/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 5.11.12 To 15.12.12 | BT | 8600 | 62 | 34 | 4 | 17 | 25 | 9 | 98 | 22 | 163 | NIL | NIL | NIL | Good |
| | | | | | AT | 8400 | 63 | 32 | 5 | 17 | 25 | 10 | 98 | 20 | 162 | NIL | NIL | NIL | |
| 32. | 1388 | Rajeshwari 38/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 5.11.12 To 12.12.12 | BT | 8800 | 58 | 37 | 5 | 12 | 20 | 11 | 100 | 22 | 159 | NIL | NIL | PCS | Moderate |
| | | | | | AT | 8500 | 60 | 36 | 4 | 12 | 20 | 11 | 98 | 18 | 158 | NIL | NIL | FPC | |
| 33. | 3550 | Suseela 64/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 6.11.12 To 8.12.12 | BT | 8600 | 61 | 34 | 5 | 10 | 24 | 12 | 110 | 19 | 180 | NIL | NIL | PCS | Good |
| | | | | | AT | 8800 | 57 | 36 | 7 | 10 | 24 | 12 | 108 | 20 | 175 | NIL | NIL | FPC | |
| 34. | 1664 | Kalavathy 38/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 6.11.12 To 13.12.12 | BT | 8800 | 58 | 37 | 5 | 9 | 18 | 13 | 100 | 21 | 190 | NIL | NIL | FPC | Good |
| | | | | | AT | 8700 | 55 | 39 | 6 | 9 | 19 | 13 | 97 | 20 | 190 | NIL | NIL | NIL | |
| 35. | 1661 | Murugesan 30/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 6.11.12 To 15.12.12 | BT | 10100 | 66 | 30 | 4 | 4 | 9 | 15 | 89 | 21 | 158 | NIL | NIL | NIL | Good |
| | | | | | AT | 10200 | 67 | 29 | 4 | 4 | 9 | 15 | 91 | 18 | 164 | NIL | NIL | NIL | |

**Table No: 4.5.8**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, OUT – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| SI NO | Op. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | |
| 36. | 1834 | Balu 40/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 7.11.12 To 14.12.12 | BT | 10900 | 64 | 31 | 5 | 4 | 26 | 13 | 108 | 20 | 163 | NIL | NIL | NIL | Satisfactory |
| | | | | | AT | 10800 | 65 | 31 | 4 | 4 | 26 | 13 | 98 | 19 | 160 | NIL | NIL | NIL | |
| 37. | 1923 | Vaithilingam 40/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 7.11.12 To 15.12.12 | BT | 10500 | 62 | 34 | 4 | 6 | 18 | 12 | 100 | 16 | 158 | NIL | NIL | PCS | Good |
| | | | | | AT | 10400 | 61 | 35 | 4 | 6 | 18 | 12 | 100 | 17 | 155 | NIL | NIL | NIL | |
| 38. | 2542 | Sanmugam 47/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 10.11.12 To 16.12.12 | BT | 9800 | 57 | 38 | 5 | 10 | 20 | 11 | 98 | 19 | 157 | NIL | NIL | PCS | Good |
| | | | | | AT | 9800 | 56 | 39 | 5 | 10 | 20 | 11 | 96 | 16 | 162 | NIL | NIL | FPC | |
| 39. | 3551 | Ranga 48/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 16.11.12 To 27.12.12 | BT | 9900 | 59 | 37 | 4 | 9 | 16 | 13 | 115 | 21 | 167 | NIL | NIL | NIL | Satisfactory |
| | | | | | AT | 10000 | 59 | 38 | 3 | 9 | 16 | 13 | 105 | 20 | 163 | NIL | NIL | NIL | |
| 40. | 3559 | Sumathi 45/Female | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 16.11.12 To 25.12.12 | BT | 9500 | 58 | 36 | 6 | 15 | 30 | 10 | 96 | 19 | 180 | NIL | NIL | NIL | Good |
| | | | | | AT | 9500 | 58 | 38 | 4 | 15 | 30 | 10 | 94 | 20 | 178 | NIL | NIL | NIL | |

**Table No: 4.5.9**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, IN – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| SI NO | IP. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 1. | 908/ 2732 | Elumalai 40/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 5.7.12 To 18.7.12 | BT | 9900 | 61 | 36 | 3 | 15 | 23 | 12 | 106 | 18 | 178 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 9900 | 60 | 37 | 3 | 15 | 23 | 12 | 102 | 20 | 174 | NIL | NIL | NIL | | |
| 2. | 1076/ 7058 | Moorthy 51/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 23.7.12 To 22.8.12 | BT | 10000 | 59 | 38 | 3 | 16 | 25 | 11.5 | 114 | 21 | 186 | NIL | NIL | PCS | - | Poor |
| | | | | | AT | 10000 | 58 | 40 | 2 | 16 | 23 | 12 | 108 | 20 | 183 | NIL | NIL | NIL | | |
| 3. | 1146/ 8782 | Kolnchi 31/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 28.7.12 To 5.8.12 | BT | 10200 | 62 | 34 | 4 | 12 | 21 | 10 | 106 | 19 | 170 | NIL | NIL | FPC | - | Satisfactory |
| | | | | | AT | 10100 | 60 | 35 | 5 | 10 | 18 | 11 | 102 | 20 | 173 | NIL | NIL | NIL | | |
| 4. | 1156/ 9029 | Premkumar 36/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 30.7.12 To 5.8.12 | BT | 9900 | 63 | 34 | 3 | 6 | 13 | 13 | 108 | 18 | 169 | NIL | NIL | FPC | - | Satisfactory |
| | | | | | AT | 9800 | 62 | 34 | 4 | 8 | 15 | 13 | 102 | 21 | 163 | NIL | NIL | NIL | | |
| 5. | 1274/ 2712 | Venkatesan 50/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 14.8.12 To 27.8.12 | BT | 9600 | 64 | 33 | 3 | 13 | 25 | 10 | 110 | 20 | 166 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 9600 | 63 | 34 | 3 | 13 | 25 | 10.5 | 106 | 20 | 160 | NIL | NIL | NIL | | |

**Table No: 4.5.10**

**CLINICAL STUDY ON *ARITHIRAADHI CHOORANAM*, IN – PATIENTS DEPT.IN THE MANAGEMENT OF KAMALAI**

| SI NO | IP. No | Name/ Age/ Sex | Complaints | Duration of Days | BT & AT | INVESTIGATION | | | | | | | | | | | | | | Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | BLOOD | | | | | | | | | Blood CL | Urine | | | X ray bms/ Endoscopy | |
| | | | | | | TC cells/cumm | DC (%) | | | ESR(mm) | | Hb gm | Sgr mg/dl | Ur mg/dl | | Sgr | Alb | Dep | | |
| | | | | | | | P | L | E | ½ hr | 1 hr | | | | | | | | | |
| 6. | 1416/ 7108 | Sasikumar 48/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 3.9.12 To 21.10.12 | BT | 9100 | 60 | 34 | 6 | 9 | 16 | 11 | 95 | 20 | 188 | NIL | NIL | PCS | - | Good |
| | | | | | AT | 9000 | 58 | 37 | 5 | 4 | 8 | 11 | 90 | 19 | 186 | NIL | NIL | NIL | | |
| 7. | 29/ 742 | Gopal 55/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 18.9.12 TO 13.10.12 | BT | 9700 | 60 | 35 | 5 | 12 | 20 | 12 | 108 | 20 | 162 | NIL | NIL | PCS | - | Poor |
| | | | | | AT | 9700 | 56 | 40 | 4 | 12 | 22 | 12 | 102 | 17 | 155 | NIL | NIL | NIL | | |
| 8. | 223/ 6409 | John aabraham 48/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 11.10.12To 22.10.12 | BT | 9900 | 58 | 35 | 7 | 5 | 15 | 13 | 110 | 20 | 170 | NIL | NIL | FPC | - | Satisfactory |
| | | | | | AT | 9600 | 56 | 38 | 6 | 5 | 15 | 13 | 105 | 18 | 162 | NIL | NIL | NIL | | |
| 9. | 323/ 9390 | Ganesh 43/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 26.10.12 To 17.12.12 | BT | 10100 | 64 | 32 | 4 | 4 | 9 | 14 | 96 | 21 | 190 | NIL | NIL | FPC | - | Satisfactory |
| | | | | | AT | 10100 | 62 | 34 | 4 | 4 | 9 | 14 | 96 | 20 | 185 | NIL | NIL | NIL | | |
| 10. | 336/ 299 | Kannan 35/Male | Anorexia, Nausea, Vomiting, Fatigue, Insomnia, Yellow colored sclera | 31.10.12 To 12.11.12 | BT | 10000 | 62 | 33 | 5 | 6 | 12 | 13.5 | 125 | 18 | 178 | NIL | NIL | NIL | - | Good |
| | | | | | AT | 10000 | 63 | 33 | 4 | 6 | 12 | 13.5 | 120 | 18 | 175 | NIL | NIL | NIL | | |

**Table No: 4.5.11**

## LIVER FUNCTION TEST

| S. No | OP No | Name/age/sex | Duration of Treatment | Duration | LIVER FUNCTION TEST | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | AST(SGOT) (IU/L) | ALT(SGPT) (IU/L) | ALP (IU/L) | Total Bilirubin (mg/dl) | Total protein g/dl | Albumin g/dl |
| 1. | 5735 | Chandran 50/ Male | 17.7.12 To 20.8.12 | BT | 95 | 93 | 130 | 2.8 | 5.7 | 4.2 |
| | | | | AT | 45 | 40 | 116 | 1.2 | 6.4 | 4.5 |
| 2. | 2355 | Vishwanathan 36/Male | 18.7.12 To 15.8.12 | BT | 50 | 48 | 72 | 1.6 | 4.6 | 3.2 |
| | | | | AT | 40 | 30 | 68 | 0.9 | 4.6 | 3.2 |
| 3. | 3875 | Sangeetha 40/Female | 21.7.12 To 25.8.12 | BT | 56 | 62 | 98 | 2.4 | 6.2 | 3.8 |
| | | | | AT | 38 | 30 | 96 | 1.2 | 7.2 | 4.1 |
| 4. | 9768 | Meganathan 39/Male | 25.7.12 To 30.8.12 | BT | 60 | 52 | 75 | 2.0 | 7.2 | 5.3 |
| | | | | AT | 39 | 32 | 70 | 1.0 | 7.4 | 5.4 |
| 5. | 3674 | Janani 37/Female | 28.7.12 To 28.8.12 | BT | 50 | 52 | 82 | 2.5 | 5.5 | 2.8 |
| | | | | AT | 35 | 47 | 78 | 1.4 | 6.2 | 3.4 |

**Table No: 4.5.12**

## LIVER FUNCTION TEST

| S. No | OP No | Name/age/sex | Duration of Treatment | Duration | LIVER FUNCTION TEST | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | AST(SGOT) (IU/L) | ALT(SGPT) (IU/L) | ALP (IU/L) | Total Bilirubin (mg/dl) | Total protein g/dl | Albumin g/dl |
| 6. | 5174 | Ramkrishnan 36/ Male | 30.7.12 To 5.9.12 | BT | 56 | 50 | 78 | 2.8 | 4.2 | 2.3 |
| | | | | AT | 26 | 25 | 76 | 1.0 | 4.4 | 2.6 |
| 7. | 9531 | Visalakshi 53/Female | 31.7.12 To 3.9.12 | BT | 58 | 56 | 82 | 2.1 | 7.1 | 4.3 |
| | | | | AT | 27 | 28 | 75 | 0.9 | 7.3 | 4.5 |
| 8. | 7105 | Vijaya 41/Female | 2.8.12 To 30.8.12 | BT | 80 | 64 | 98 | 1.2 | 6.2 | 3.6 |
| | | | | AT | 25 | 30 | 92 | 0.7 | 6.4 | 3.8 |
| 9. | 7219 | Siraj 37/Female | 4.8.12 To 10.9.12 | BT | 57 | 50 | 76 | 3.2 | 5.5 | 2.8 |
| | | | | AT | 42 | 40 | 74 | 1.2 | 6.8 | 3.9 |
| 10. | 8593 | Devi ammal 39/Female | 5.8.12 To 9.9.12 | BT | 56 | 50 | 90 | 2.8 | 7.2 | 4.5 |
| | | | | AT | 38 | 42 | 88 | 0.8 | 7.4 | 4.8 |

**Table No: 4.5.13**

## LIVER FUNCTION TEST

| S. No | OP No | Name/age/sex | Duration of Treatment | Duration | LIVER FUNCTION TEST | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | AST(SGOT) (IU/L) | ALT(SGPT) (IU/L) | ALP (IU/L) | Total Bilirubin (mg/dl) | Total protein g/dl | Albumin g/dl |
| 11. | 6897 | Kamaraj 41/Male | 7.8.12 To 14.9.12 | BT | 57 | 66 | 128 | 2.0 | 5.8 | 3.8 |
| | | | | AT | 31 | 38 | 112 | 1.1 | 5.8 | 4.8 |
| 12. | 2309 | Jayaprasath 37/Male | 10.8.12 To 18.9.12 | BT | 56 | 50 | 80 | 2.3 | 5.2 | 2.8 |
| | | | | AT | 29 | 31 | 72 | 1.0 | 6.2 | 3.8 |
| 13. | 8909 | Arokiya raj 39/Male | 11.8.12 To 19.9.12 | BT | 58 | 44 | 98 | 2.6 | 6.2 | 3.2 |
| | | | | AT | 46 | 42 | 96 | 1.2 | 6.3 | 3.3 |
| 14. | 214 | Jeevanandham 34/Male | 13.8.12 To 14.9.12 | BT | 87 | 70 | 100 | 2.5 | 5.8 | 3.8 |
| | | | | AT | 46 | 34 | 94 | 1.3 | 6.4 | 4.1 |
| 15. | 136 | Puhpa 36/Female | 16.8.12 To 20.9.12 | BT | 56 | 47 | 106 | 2.7 | 6.5 | 3.8 |
| | | | | AT | 46 | 34 | 100 | 1.4 | 7.4 | 4.8 |

**Table No: 4.5.14**

## LIVER FUNCTION TEST

| S. No | OP No | Name/age/sex | Duration of Treatment | Duration | LIVER FUNCTION TEST | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | AST(SGOT) (IU/L) | ALT(SGPT) (IU/L) | ALP (IU/L) | Total Bilirubin (mg/dl) | Total protein g/dl | Albumin g/dl |
| 16. | 391 | Valarmathi 42/Female | 20.8.12 To 21.9.12 | BT | 54 | 46 | 95 | 3.0 | 6.1 | 4.2 |
| | | | | AT | 28 | 22 | 92 | 1.2 | 5.6 | 4.2 |
| 17. | 1469 | Prithiviraj 39/Male | 22.8.12 To 26.9.12 | BT | 160 | 143 | 109 | 2.8 | 7.4 | 4.8 |
| | | | | AT | 68 | 73 | 97 | 1.0 | 7.4 | 5.1 |
| 18. | 1503 | Krishnaveni 34/Female | 24.8.12 To 30.8.12 | BT | 210 | 180 | 135 | 1.8 | 6.2 | 3.5 |
| | | | | AT | 66 | 70 | 120 | 0.9 | 6.4 | 3.6 |
| 19. | 205 | Rejina mary 26/Female | 26.8.12 To 29.9.12 | BT | 190 | 178 | 135 | 1.5 | 7.2 | 3.8 |
| | | | | AT | 60 | 51 | 88 | 0.8 | 6.6 | 3.9 |
| 20. | 7542 | Aarumugam 51/Male | 29.8.12 To 5.10.12 | BT | 130 | 112 | 132 | 1.8 | 7.6 | 3.9 |
| | | | | AT | 60 | 56 | 116 | 0.8 | 7.4 | 3.9 |

**Table No: 4.5.15**

## LIVER FUNCTION TEST

| S. No | OP No | Name/age/sex | Duration of Treatment | Duration | LIVER FUNCTION TEST | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | AST(SGOT) (IU/L) | ALT(SGPT) (IU/L) | ALP (IU/L) | Total Bilirubin (mg/dl) | Total protein g/dl | Albumin g/dl |
| 21. | 3841 | Kalaiselvi 36/Female | 8.9.12 To 15.10.12 | BT | 120 | 140 | 108 | 1.8 | 8.2 | 5.0 |
| | | | | AT | 48 | 53 | 104 | 1.1 | 7.8 | 5.0 |
| 22. | 8444 | Jothimani 44/Male | 15.9.12 To 25.10.12 | BT | 166 | 152 | 106 | 2.0 | 5.4 | 4.1 |
| | | | | AT | 52 | 50 | 118 | 1.0 | 5.8 | 4.4 |
| 23. | 5870 | Siva Arvinth 40/Male | 26.9.12 To 26.10.12 | BT | 120 | 158 | 135 | 2.6 | 6.0 | 4.3 |
| | | | | AT | 30 | 36 | 117 | 0.9 | 6.7 | 4.5 |
| 24. | 5086 | Kanagavalli 50/Female | 16.10.12 To 15.11.12 | BT | 147 | 93 | 127 | 1.5 | 5.4 | 3.2 |
| | | | | AT | 41 | 44 | 112 | 0.9 | 6.3 | 4.1 |
| 25. | 8010 | Meenakshi 30/Female | 18.10.12 To 30.12.12 | BT | 95 | 50 | 140 | 1.7 | 5.8 | 3.5 |
| | | | | AT | 34 | 22 | 106 | 1.3 | 5.1 | 3.4 |

**Table No: 4.5.16**

## LIVER FUNCTION TEST

| S. No | OP No | Name/age/sex | Duration of Treatment | Duration | LIVER FUNCTION TEST | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | AST(SGOT) (IU/L) | ALT(SGPT) (IU/L) | ALP (IU/L) | Total Bilirubin (mg/dl) | Total protein g/dl | Albumin g/dl |
| 26. | 225 | Munusamy 50/Male | 30.10.12 To 18.12.12 | BT | 145 | 85 | 130 | 3.5 | 6.8 | 4.6 |
| | | | | AT | 48 | 38 | 94 | 0.9 | 7.4 | 4.3 |
| 27. | 318 | Mallika 38/Female | 31.10.12 To 04.12.12 | BT | 170 | 92 | 110 | 2.1 | 6.3 | 3.8 |
| | | | | AT | 52 | 36 | 97 | 1.0 | 6.4 | 3.9 |
| 28. | 947 | Naveen kumar 35/Male | 3.11.12 To 6.12.12 | BT | 175 | 125 | 118 | 1.2 | 5.2 | 2.6 |
| | | | | AT | 60 | 48 | 82 | 0.8 | 7.0 | 3.4 |
| 29. | 948 | Murugan 38/Male | 3.11.2012 To 10.12.12 | BT | 194 | 132 | 112 | 1.4 | 6.5 | 4.1 |
| | | | | AT | 72 | 60 | 94 | 0.9 | 6.9 | 4.3 |
| 30. | 1379 | Vasanthi 30/Female | 05.11.12 To 6.12.12 | BT | 175 | 132 | 101 | 1.2 | 5.3 | 4.5 |
| | | | | AT | 69 | 52 | 98 | 0.9 | 6.2 | 4.9 |

**Table No: 4.5.17**

## LIVER FUNCTION TEST

| S. No | OP No | Name/age/sex | Duration of Treatment | Duration | LIVER FUNCTION TEST | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | AST(SGOT) (IU/L) | ALT(SGPT) (IU/L) | ALP (IU/L) | Total Bilirubin (mg/dl) | Total protein g/dl | Albumin g/dl |
| 31. | 1973 | Daisy 30/Female | 5.11.12 To 15.12.12 | BT | 170 | 92 | 110 | 2.1 | 6.3 | 3.8 |
| | | | | AT | 62 | 36 | 97 | 1.0 | 6.4 | 3.9 |
| 32. | 1388 | Rajeshwari 38/Female | 5.11.12 To 12.12.12 | BT | 120 | 140 | 108 | 1.8 | 8.2 | 5.0 |
| | | | | AT | 48 | 53 | 104 | 1.1 | 7.8 | 5.0 |
| 33. | 3550 | Suseela 64/Female | 6.11.12 To 8.12.12 | BT | 140 | 70 | 90.3 | 1.2 | 6.8 | 4.2 |
| | | | | AT | 28 | 35 | 82 | 0.8 | 6.9 | 4.3 |
| 34. | 1664 | Kalavathy 38/Male | 6.11.12 To 13.12.12 | BT | 110 | 88 | 126 | 1.3 | 6.5 | 4.7 |
| | | | | AT | 52 | 40 | 116 | 0.7 | 6.5 | 4.7 |
| 35. | 1661 | Murugesan 30/Male | 6.11.12 To 15.12.12 | BT | 102 | 78 | 78 | 1.5 | 5.2 | 2.6 |
| | | | | AT | 44 | 53 | 54 | 0.7 | 6.4 | 3.8 |

## LIVER FUNCTION TEST

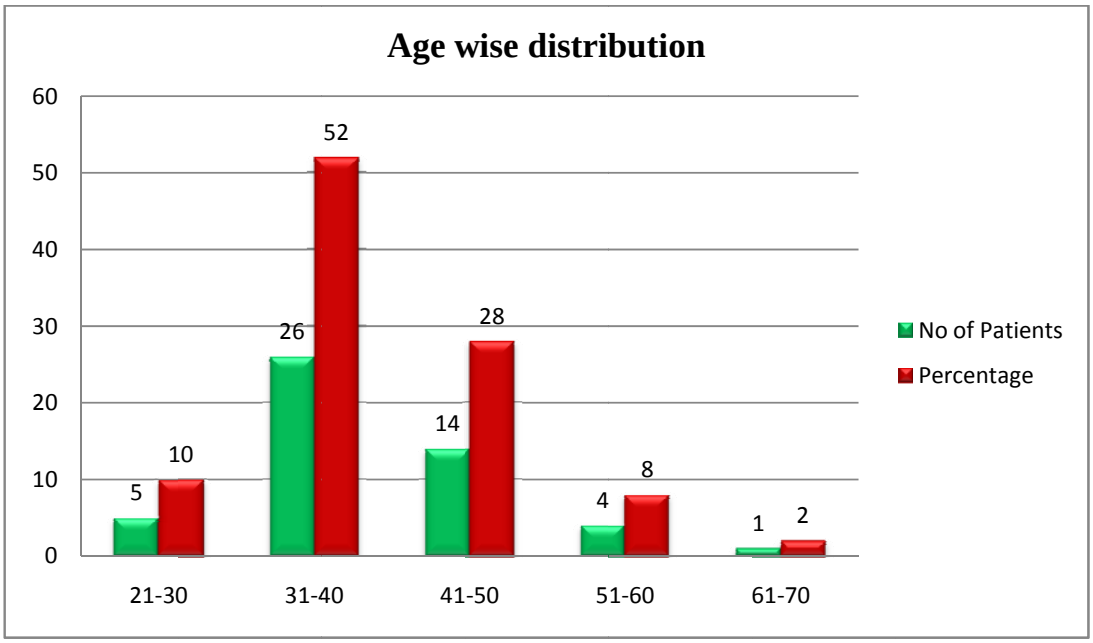
**Table No: 4.5.18**

| S. No | OP No | Name/age/sex | Duration of Treatment | Duration | LIVER FUNCTION TEST | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | AST(SGOT) (IU/L) | ALT(SGPT) (IU/L) | ALP (IU/L) | Total Bilirubin (mg/dl) | Total protein g/dl | Albumin g/dl |
| 36. | 1834 | Balu 40/Male | 7.11.12 To 14.12.12 | BT | 95 | 93 | 130 | 2.8 | 5.7 | 4.2 |
| | | | | AT | 45 | 40 | 116 | 1.2 | 6.4 | 4.5 |
| 37. | 1923 | Vaithilingam 40/Male | 7.11.12 To 15.12.12 | BT | 210 | 180 | 135 | 1.8 | 6.2 | 3.5 |
| | | | | AT | 66 | 70 | 120 | 0.9 | 6.4 | 3.6 |
| 38. | 2542 | Sanmugam 47/Male | 10.11.12 To 16.12.12 | BT | 190 | 177 | 135 | 1.5 | 7.2 | 3.8 |
| | | | | AT | 60 | 54 | 89 | 0.8 | 6.6 | 3.9 |
| 39. | 3551 | Ranga 48/Male | 16.11.12 To 27.12.12 | BT | 112 | 112 | 139 | 1.8 | 7.6 | 3.9 |
| | | | | AT | 59 | 56 | 116 | 0.9 | 7.4 | 4.2 |
| 40. | 3559 | Sumathi 45/Female | 16.11.12 To 25.12.12 | BT | 124.8 | 84.8 | 152.6 | 1.67 | 7.2 | 5.2 |
| | | | | AT | 43.2 | 32.7 | 112.6 | 0.9 | 7.4 | 5.3 |

**Table No: 4.5.19**

## Age wise distribution

| SL. NO | AGE IN YEARS | NO. OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | 21-30 | 5 | 10 |
| 2 | 31-40 | 26 | 52 |
| 3 | 41-50 | 14 | 28 |
| 4 | 51-60 | 4 | 8 |
| 5 | 61-70 | 1 | 2 |
| TOTAL | | 50 | 100 |




**Inference:**

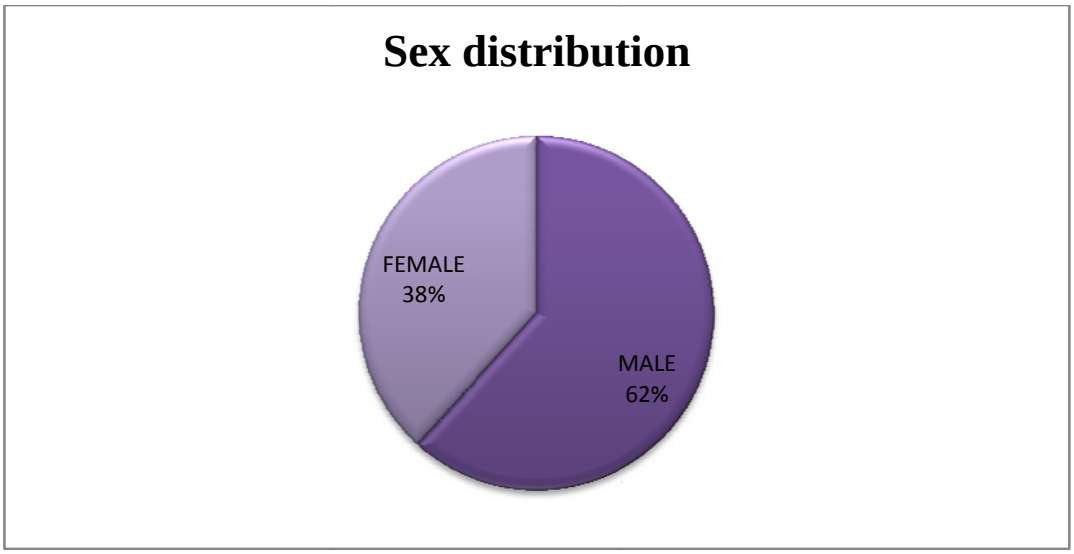
Among 50 patients,

- 5 patient belongs to the age group of 21-30 years
- 26 patients belongs to the age group of 31-40 years
- 14 patients belongs to the age group of 41-50 years
- 4 patients belongs to the age group of 51-60 years
- 1 patient belongs to the age group of 61-70 years

**Table No: 4.5.20**

## Sex distribution

| SL. NO | SEX | NO. OF PATIENTS | PERCENTAGE |
|---|---|---|---|
| 1 | Male | 29 | 58 |
| 2 | Female | 21 | 42 |
| TOTAL | | 50 | 100 |




## Inference:

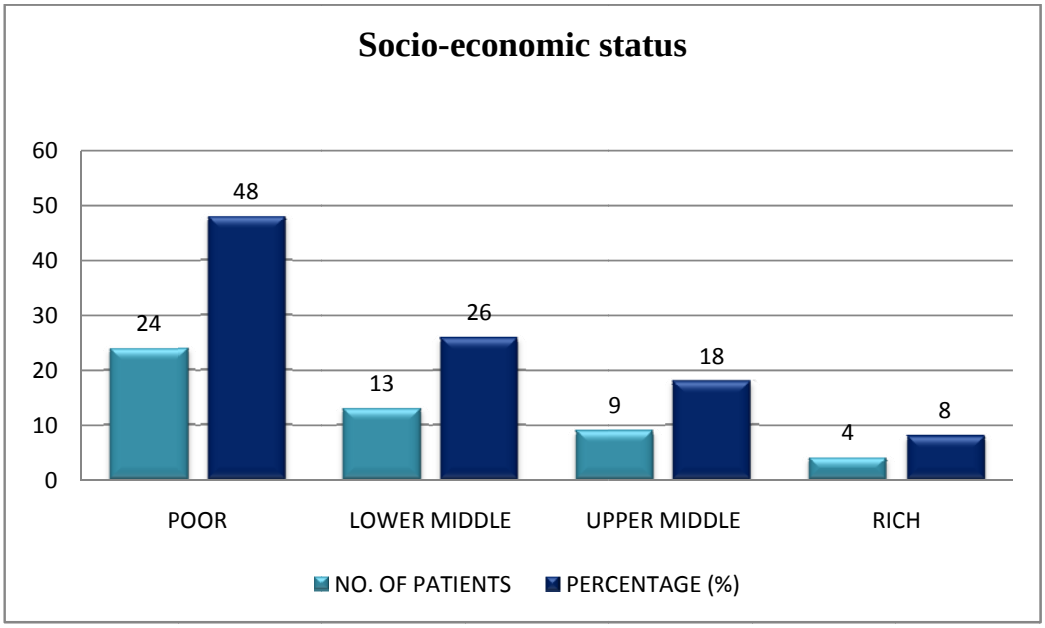
Among 50 patients,

- 29 patients were male
- 21 patients were female

**Table No: 4.5.21**

## Socio-economic status

| SL. NO | SOCIO – ECONOMIC STATUS | NO. OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | Poor | 24 | 48 |
| 2 | Lower middle | 13 | 26 |
| 3 | Upper middle | 9 | 18 |
| 4 | Rich | 4 | 8 |
| TOTAL | | 50 | 100 |




**Inference:**

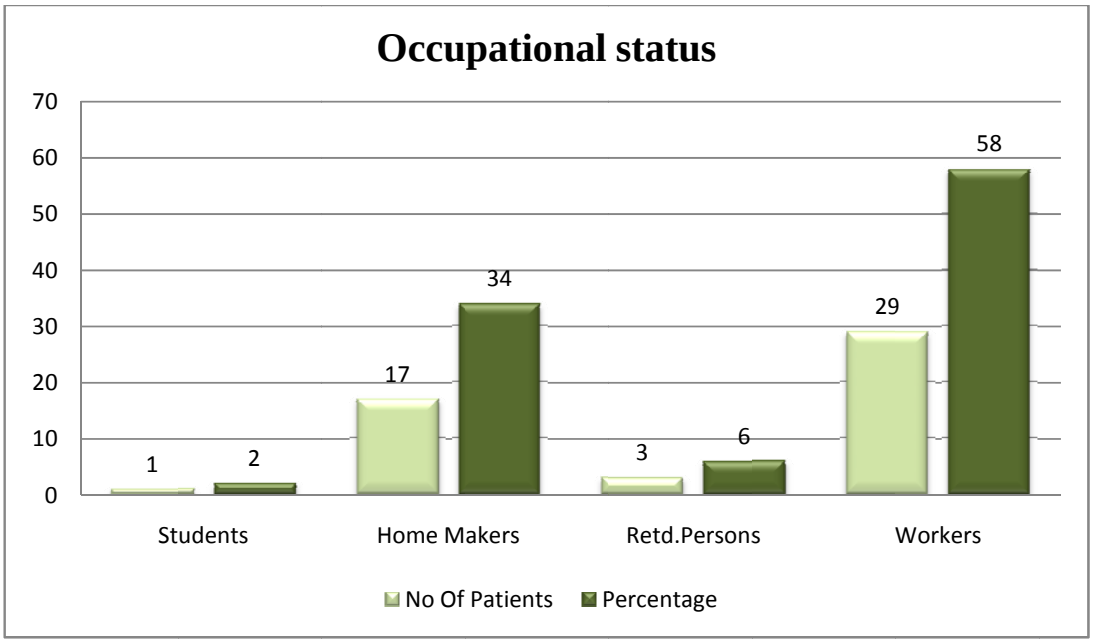
Among 50 patients,

- 24 patients were poor.
- 13 patients were lower-middle.
- 9 patients were upper middle.
- 4 patients were rich.

**Table No: 4.5.22**

## Occupational status

| SL. NO | OCCUPATION | NO. OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | Students | 1 | 2 |
| 2 | Home makers | 17 | 34 |
| 3 | Retired persons | 3 | 6 |
| 4 | Workers | 29 | 58 |
| TOTAL | | 50 | 100 |




**Inference:**

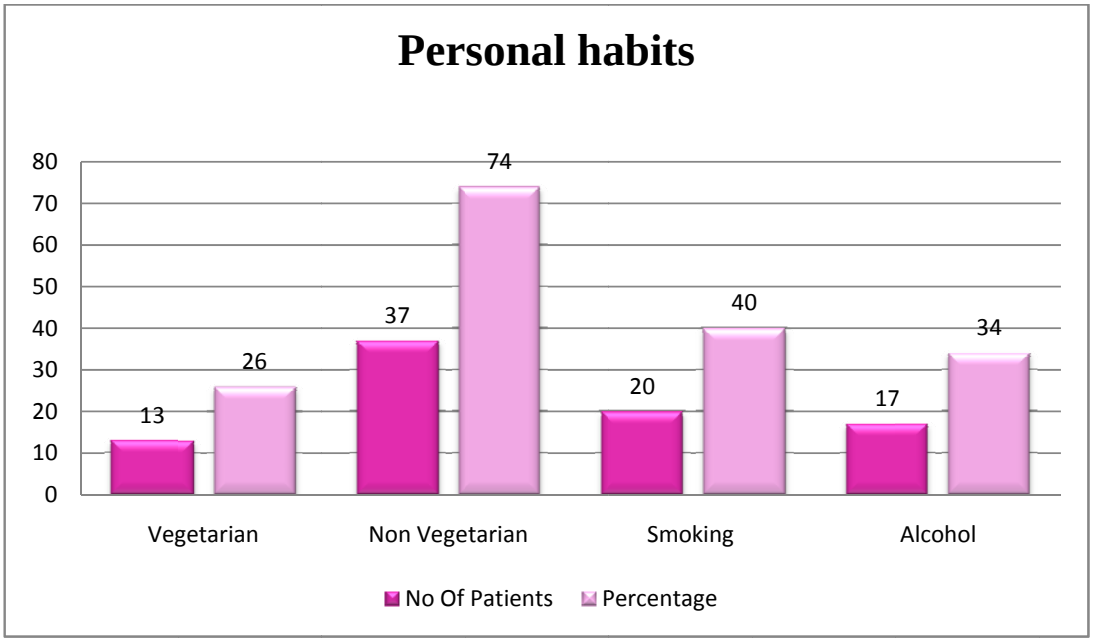
Among 50 patients,

- 1 (2%) patients were Students.
- 17 (34%) patients were Home makers.
- 3 (6%) patients were Retired persons.
- 29 (58%) patients were Workers.

**Table No: 4.5.23**

## Personal habits

| SL. NO | PERSONAL HABITS | NO. OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | Vegetarian | 13 | 26 |
| 2 | Non-vegetarian | 37 | 74 |
| 3 | Smoking | 20 | 40 |
| 4 | Alcohol | 17 | 34 |




**Inference:**

Among 50 patients,

- 13 Patients were Vegetarian
- 37 Patients were Non Vegetarian
- 20 Patients were Smokers
- 17 Patients were Drinkers

# 5. RESULTS AND DISCUSSION

### 4.2.1. Physico-chemical analysis:

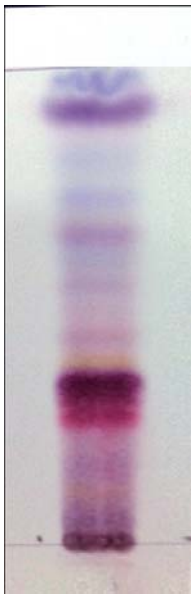
**Report of *arithradi chooranam***

**Table No: 4.2.1.1**

| S.No | Parameter | Mean Value |
|---|---|---|
| 1. | Loss on Drying at 105°C | 7.355 % |
| 2. | Total Ash | 17.584 % |
| 3. | Acid insoluble Ash | 0.096 % |
| 4. | Water Soluble Extractive | 45.40 % |
| 5. | Alcohol Soluble Extractive | 39.30 % |
| 6. | pH | 6.0 |

**TLC result of *arithiraadhi chooranam***

**Figure No: 4.2.1.2**



After spray with visualizing agent

**Table No: 4.2.1.2**

| Sl.No | After Dipping in Vanillin-Sulphuric acid | |
|---|---|---|
| | **Rf value** | **Colour of the spot** |
| 1 | 0.06 | Purple |
| 2 | 0.11 | Yellow |
| 3 | 0.17 | Purple |
| 4 | 0.28 | Pink |
| 5 | 0.34 | Majenta |
| 6 | 0.39 | Yellow |
| 7 | 0.44 | Pink |
| 8 | 0.55 | Pink |
| 11 | 0.66 | Purple |
| 12 | 0.74 | Purple |
| 13 | 0.82 | Purple |
| 14 | 0.91 | Purple |

## 4.2.4 Qualitative phytochemical analysis:

## Result of *arithiraadhi chooranam*

**Table No: 4.2.4**

| Qualitative Phytochemical Tests | | |
|---|---|---|
| 1. | Alkaloids | - ve |
| 2. | Anthraquinones | + ve |
| 3. | Triterpenes | + ve |
| 4. | Flavonoids | + ve |
| 5. | Steroids | + ve |
| 6. | Phenol | + ve |
| 7. | Tannin | + ve |

Phytochemical analysis of *Arithiraadhi chooranam* showed the following,

Presence of Anthraquinone, Triterpenes, Flavonoids, Steroids, Phenol and Tannins.

*Curcuma longa* which is one of the ingredients in the *Arithiraadhi chooranam*, protects the structural integrity of hepatocyte membrane. ( Somchit et al- 2005 )

Curcumin which is one of the phyto chemical constituents of *Curcuma longa* also has effect on the cellular oxidative metabolism, which may improve the hepatoprotective mechanism. (Donatus et al – 1998 )

Tannins and flavanoids of *Phyllanthus emblica* contain anti oxidant and hepatoprotective properties. ( Kawala et al- 2002 )

Tannins, flavanoids and other phytochemicals which are present in *Arithiraadhi chooranam* may be responsible for its Hepatoprotective activity.

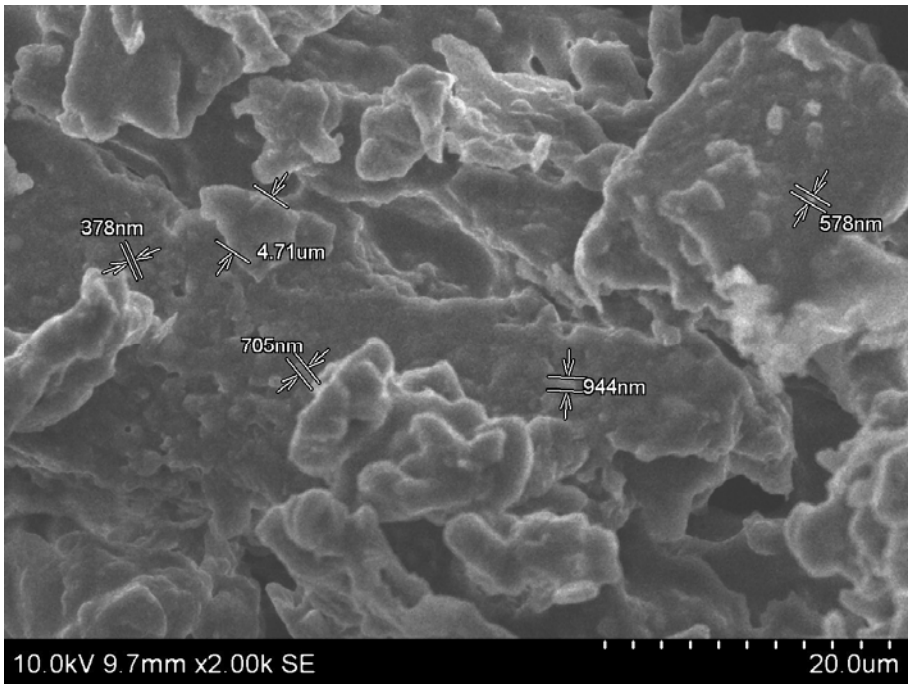
### 4.2.1.3 Fourier transform infrared spectroscopy (FTIR)

**Ftir result of *arithiraadhi chooranam***

| **Peak** | **Functional group** |
|---|---|
| 3434 $cm^{-1}$ | Alcohol phenol O-H stretch |
| 2920 $cm^{-1}$ | Carboxylic acid O-H stretch |
| 2852 $cm^{-1}$ | Carboxylic acid O-H stretch |
| 2129 $cm^{-1}$ | Alkynyl C=C stretch |
| 1633 $cm^{-1}$ | Amide C=O stretch |
| 1379 $cm^{-1}$ | Alkyl methyl |
| 1233 $cm^{-1}$ | C-O ether aromatic |
| 1163 $cm^{-1}$ | Esters |
| 1036 $cm^{-1}$ | Aliphatic amines |
| 667 $cm^{-1}$ | Chloro alkanes |

## 4.2.1.4 Scanning electron microscope (SEM):

**Figure No: 4.2.1.4**




## Results:

SEM picture shows Nano particle (Micro level) size of the sample.

Physical properties of known elements and materials can change as their surface to area ratio is dramatically increased, i.e. when nanoscale sizes are achieved. These changes do not take place when going from macro to micro scale. Changes in physical properties such as colloidal properties, solubility and catalytic capacity have been found very useful in areas of bioremediation and drug delivery. The extremely small size of nanoparticles allows them to penetrate cells and interact with cellular molecules. Due to nanoparticle size a low dose of the drug can cure the diseases.

### 4.2.5.Chemical analysis of *arithiraadhi chooranam:*

## Results:

The chemical analysis of *Arithiraadhi chooranam* showed the following chemicals,

Presence of Reducing sugar, Starch, Proteins, Amino acid, Sulphate, Chloride, Iron, and Tannin.

### 4.3 .Toxicological study

**Results and discussion:**

All the animals from control and all the treated dose groups up to 500 mg/kg survived throughout the dosing period of 28 days. No signs of major or significant intoxication were observed in animals from lower to higher dose groups during the dosing period of 28 days. Animals from all the treated dose groups exhibited comparable body weight gain with that of controls throughout the dosing period of 28 days. Food consumption of control and treated animals was found to be comparable throughout the dosing period of 28days. Ophthalmoscopic examination, conducted prior to and at the end of dosing period on animals from control and all the treated dose groups did not reveal any abnormality.

Haematological analysis conducted at the end of the dosing period on day 28, revealed no significant abnormalities attributable to the treatment. Biochemical analysis conducted at the end of the dosing period on day 28, revealed no remarkable abnormalities attributable to the treatment. Functional observation tests conducted at termination revealed no abnormalities. Urine analysis, conducted at the end of the dosing period in week 4 and at the end of recovery period in week 6, revealed no abnormality attributable to the treatment. Organ weight data of animals sacrificed at the end of the dosing period was found to be comparable with that of respective controls. Gross pathological examination did not reveal any abnormality. Histopathological examination did not reveal any abnormality.

**Table No: 4.3.1 Dose finding experiment and its behavioral Signs of Toxicity**

| No | Dose mg/kg | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 2000 | + | - | - | + | - | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| 2 | 5000 | + | - | - | + | - | + | - | - | - | - | - | - | - | - | - | - | - | - | - | - |

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7.Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12.Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

**Table No: 4.3.2 Body wt (g) of rats exposed to *Arithirathi Chooranam* for 28days.**

| Dose (mg/kg/day) | Days | | | | |
|---|---|---|---|---|---|
| | 1 | 7 | 14 | 21 | 28 |
| Control | 122.12±3.14 | 128.52±4.19 | 130.18±4.66 | 134.48±4.25 | 135.22±4.08 |
| 100 | 125.30±4.06 | 126.38±5.14 | 131.10±5.12 | 135.00±5.21 | 136.10±4.25 |
| 250 | 120.13±4.20 | 124.05±4.28 | 128.04±5.15 | 130.21±5.04 | 132.16±4.23 |
| 500 | 126.12±3.28 | 128.30±5.00 | 132.12±5.10 | 134.23±4.26 | 136.22±4.62 |

Values are mean ± S.E.M. (Dunnett's test). $^{ns}P>0.05$. N=6.

**Table No: 4.3.3 Food (g/day) intake of rats exposed to *Arithirathi Chooranam* for 28days.**

| Dose (mg/kg/day) | Days (gms/rats) | | | | |
|---|---|---|---|---|---|
| | 1 | 7 | 14 | 21 | 28 |
| Control | 40.25±2.13 | 42.11±2.27 | 44.57±2.42 | 42.18±2.52 | 45.64±2.58 |
| 100 | 42.12±2.26 | 45.45±2.86 | 45.55±2.20 | 44.28±2.40 | 44.51±3.44 |
| 250 | 47.20±2.28 | 46.41±2.94 | 46.34±2.14 | 45.53±2.11 | 45.46±3.51 |
| 500 | 42.32±2.55 | 47.50±2.65 | 42.28±2.55 | 45.33±2.68 | 45.55±2.29 |

**Values are mean ± S.E.M. (Dunnet's test). $^{ns}$P>0.05. N=6.**

**Table No: 4.3.4 Water (ml/day) intake of rats exposed to *Arithirathi Chooranam*.**

| Dose (mg/kg/day) | Days(ml/rat) | | | | |
|---|---|---|---|---|---|
| | 1 | 7 | 14 | 21 | 28 |
| Control | 45.12±2.18 | 50.11±3.45 | 50.11±3.28 | 50.22±3.68 | 52.12±3.81 |
| 100 | 45.18±2.26 | 52.21±3.00 | 52.18±3.45 | 56.14±3.20* | 54.25±2.62 |
| 250 | 50.22±2.28 | 50.10±3.55 | 54.24±3.60 | 54.14±2.46 | 54.42±3.42 |
| 500 | 50.21±3.45 | 55.40±3.25 | 55.00±3.27 | 48.26±3.63 | 52.08±3.34 |

**Values are mean ± S.E.M. (Dunnet't' test). *P<0.05. N=6.**

**Table No: 4.3.5 Hematological parameters after 28days treatment with *Arithirathi Chooranam* in rats.**

| Parameter | Control | 100 mg/kg | 250 mg/kg | 500 mg/kg |
|---|---|---|---|---|
| **Red blood cell ($mm^3$)** | 5.00±0.42 | 4.80±0.50 | 5.18±0.48 | 5.21±0.44 |
| **HB (%)** | 15.12±0.34 | 15.41±0.36 | 15.04±0.34 | 15.12±0.41 |
| **Leukocyte ($x10^3$/Cu.mm)** | 8.24±1.10 | 8.21±0.90 | 8.18±1.12 | 8.14±1.22 |
| **Platelets(K/µl)** | 455±24.48 | 494±30.24 | 492±30.10 | 490±30.14 |
| **MCV (gl)** | 52.56±4.18 | 50.22±4.45 | 52.11±4.26 | 51.42±4.31 |
| **N** | 14.55±2.42 | 14.62±2.12 | 14.74±2.82 | 14.82±2.23 |
| **L** | 80.30±4.28 | 82.13±4.11 | 81.62±3.84 | 82.02±3.92 |
| **M** | 1.40±0.30 | 1.29±0.31 | 1.38±0.24 | 1.44±0.22 |
| **E** | 1.04±0.11 | 1.07±0.28 | 1.00±0.14 | 1.00±0.16 |
| **B** | 0±0.00 | 0±0.00 | 0±0.00 | 0±0.00 |
| **ESR(mm)** | 1±00 | 1±00 | 1±00 | 1±00 |
| **PCV** | 44.52±2.39 | 45.24±2.41 | 45.29±2.33 | 45.18±3.42 |

**Values are mean ± S.E.M. (Dunnet't' test). nsP>0.01. N=6.**

**Table No:4.3.6 Effect of treatment with *Arithirathi Chooranam (* biochemical parameters)**

| Dose (mg/kg) | Control | 100 mg/kg | 250 mg/kg | 500 mg/kg |
|---|---|---|---|---|
| **Total Bilirubin (mg/dL)** | 0.28±0.06 | 0.28±0.05 | 0.27±0.04 | 0.28±0.03 |
| **Bilirubin direct (mg/dL)** | 0.22±0.06 | 0.22±0.07 | 0.18±0.04 | 0.20±0.04 |
| **ALP (U/L)** | 102.26±8.02 | 106.10±10.00 | 105.10±7.55 | 106.1±8.02 |
| **SGOT (U/L)** | 120.13±5.00 | 122.30±4.71 | 124.12±5.02 | 125.18±5.10 |
| **SGPT(U/L)** | 35.11±2.10 | 35.54±2.04 | 35.10±2.15 | 36.00±2.00 |
| **Total Protein(g/dl)** | 6.00±1.21 | 6.14±0.26 | 6.35±0.22 | 7.00±0.31 |
| **Albumin(g/dl)** | 2.40±0.20 | 2.43±0.24 | 3.31±0.21 | 3.10±0.18 |
| **Globulin(g/dl)** | 4.31±0.14 | 4.55±0.12 | 4.21±0.15 | 4.14±0.13 |

**Values are mean ± S.E.M. $^{ns}$P>0.05 *V*s Control N=6.**

**Table No: 4.3.7 RFT**

| Dose (mg/kg) | Control | 100 mg/kg | 250 mg/kg | 500 mg/kg |
|---|---|---|---|---|
| **Urea(mg/dL)** | 5.33±1.99 | 5.24±2.28 | 5.32±2.20 | 5.00±1.56 |
| **Creatinine (mg/dL)** | 0.71±0.05 | 0.70±0.05 | 0.70±0.05 | 0.72±0.06 |
| **Uric acid (mg/dL)** | 4.36±0.21 | 4.30±0.26 | 4.23±0.22 | 4.12±0.21 |
| **Na m.mol** | 113.12±5.00 | 112.00±4.41 | 111.22±4.00 | 112.10±3.88 |
| **K m.mol** | 5.00±2.04 | 5.14±1.76 | 5.32±1.62 | 5.11±2.21 |
| **Cl m.mol** | 102.45±4.21 | 100.00±4.62 | 101.12±4.18 | 100.33±4.04 |

**Values are mean ± S.E.M. $^{ns}$P>0.05. Vs. control group N=6.**

**Table No:4.3.8 Lipid Profile**

| Dose (mg/kg) | Control | 100 mg/kg | 250 mg/kg | 500 mg/kg |
|---|---|---|---|---|
| **Total cholesterol (mg/dL)** | 76.22±2.66 | 75.10±2.72 | 74.72±2.56 | 75.21±2.08 |
| **HDL (mg/dL)** | 118.75±2.17 | 119.52±2.55 | 121.14±3.00 | 120.24±2.20 |
| **LDL (mg/dL)** | 42.00±2.44 | 42.74±2.85 | 41.55±2.18 | 42.00±3.00 |
| **VLDL (mg/dl)** | 26.16±2.33 | 26.18±2.24 | 26.10±2.34 | 26.62±2.41 |
| **Triglycerides (mg/dl)** | 26.10±2.14 | 25.86±2.52 | 26.00±3.00 | 28.12±2.53 |
| **Blood glucose (mg/dl)** | 89.14±4.33 | 90.41±4.61 | 91.10±3.22 | 90.14±2.74 |

**Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}$P>0.01 Vs Control N=6.**

**Table No: 4.3.9 Urine Analysis**

| Parameters | Control | 100 mg/kg | 250 mg/kg | 500 mg/kg |
|---|---|---|---|---|
| **Colour** | Yellow | Yellow | Yellow | Yellow |
| **Transparency** | Clear | Slightly turbid | Slightly cloudy | Slightly turbid |
| **Specific gravity** | 1.010 | 1.010 | 1.010 | 1.010 |
| **PH** | >7.2 | >8.0 | >7.5 | >7.5 |
| **Protein** | Nil | 1+ | 1+ | 2+ |
| **Glucose** | Nil | Nil | Nil | Trace |
| **Bilirubin** | -ve | -ve | -ve | -ve |
| **Ketones** | -ve | -ve | -ve | -ve |
| **Blood** | Absent | Absent | Absent | Absent |
| **Urobilinogen** | Normal | Normal | Normal | Normal |
| **Pus cells** | 0-cells/HPF | 1-cell/HPF | 2-cells/HPF | 1-cell/HPF |
| **RBCs** | Nil | Nil | 0-1cells/HPF | Nil |
| **Epithelial cells** | Nil | 1-cell/HPF | Nil | 1-cell/HPF |
| **Crystals** | Nil | Nil | Nil | Nil |
| **Casts** | Nil | Nil | Nil | Nil |

**Table No:4.3.10 Effect of oral administration of *Arithirathi Chooranam* on organ weight**

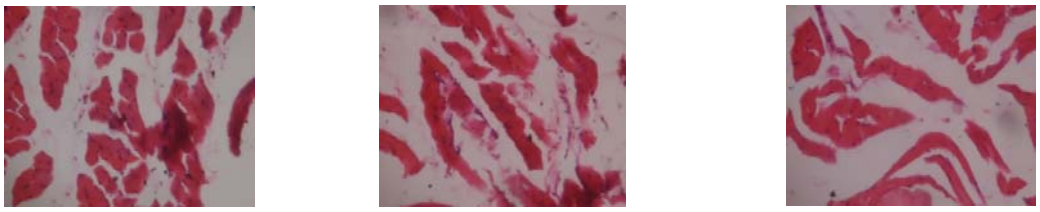
| Dose (mg/kg) | Control | 100 mg/kg | 250 mg/kg | 500 mg/kg |
|---|---|---|---|---|
| **Liver (g)** | 3.10±0.18 | 3.14±0.16 | 3.12±0.14 | 3.10±0.10 |
| **Heart (g)** | 0.32±0.06 | 0.34±0.05 | 0.35±0.06 | 0.34±0.04 |
| **Lung (g)** | 0.44±0.15 | 0.45±0.10 | 0.44±0.15 | 0.45±0.16 |
| **Spleen (g)** | 0.42±0.06 | 0.44±0.05 | 0.45±0.05 | 0.46±0.05 |
| **Ovary (g)** | 1.28±0.15 | 1.25±0.10 | 1.23±0.15 | 1.22±0.15 |
| **Testes (g)** | 2.10±0.16 | 2.20±0.12 | 2.20±0.21 | 2.21±0.14 |
| **Brain (g)** | 2.00±0.12 | 2.02±0.14 | 2.00±0.13 | 2.02±0.12 |
| **Kidney (g)** | 0.85±0.04 | 0.84±0.05 | 0.85±0.05 | 0.86±0.05 |
| **Stomach (g)** | 1.16±0.12 | 1.12±0.10 | 1.12±0.12 | 1.12±0.10 |

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}$P>0.01 Vs Control N=6.

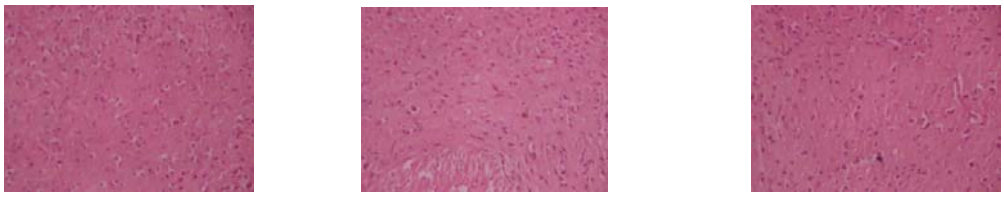
## SUB ACUTE TOXICITY STUDY

### Histopathological features

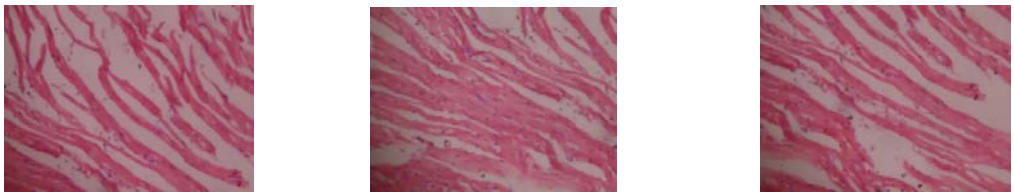
**100mg** **250mg** **500mg**



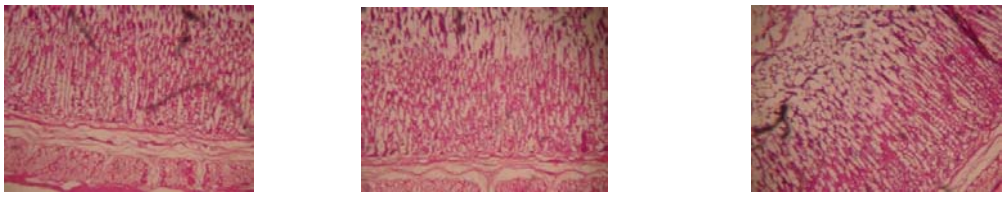
**Bone – Figure No: 4.3.1**



**Brain – Figure No: 4.3.2**

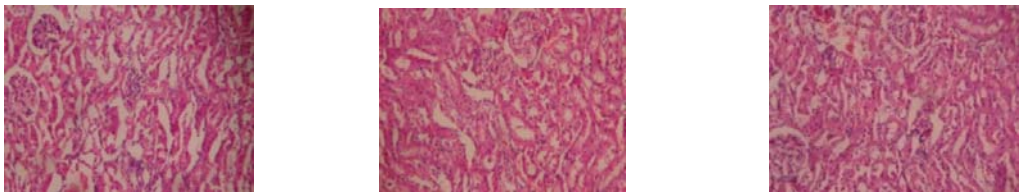


**Heart – Figure No: 4.3.3**

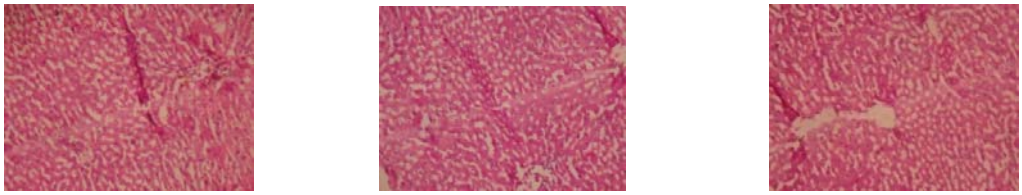


**Intestine – Figure No: 4.3.4**

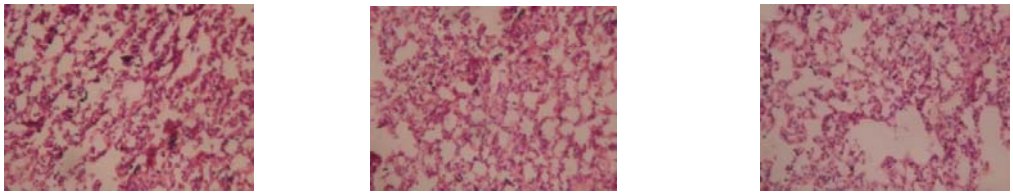
**100mg** **250mg** **500mg**



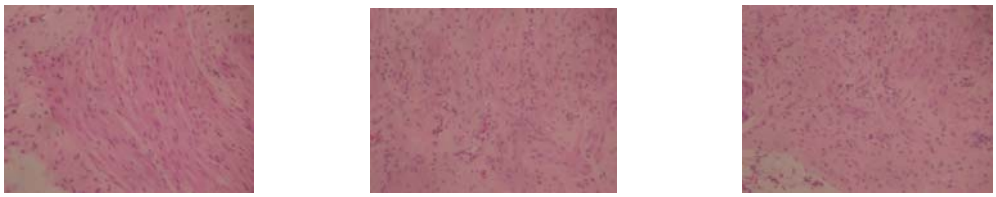
**Kidney – Figure No: 4.3.5**



**Liver – Figure No: 4.3.6**

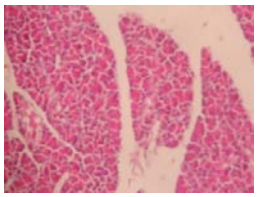
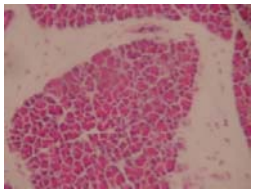
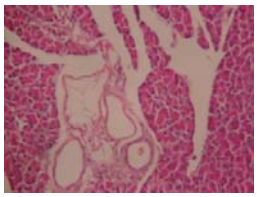


**Lung – Figure No: 4.5.7**

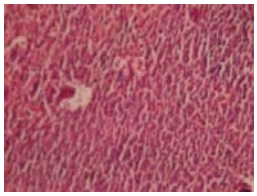
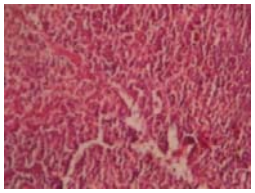
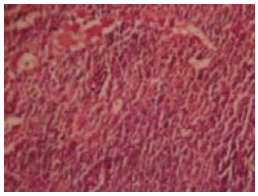


**Ovary – Figure No: 4.3.8**

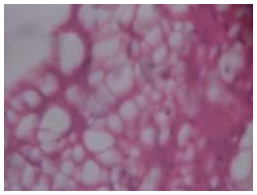
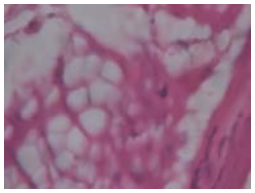
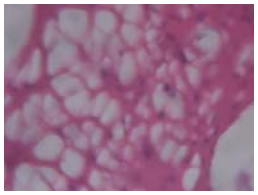
**100mg** **250mg** **500mg**

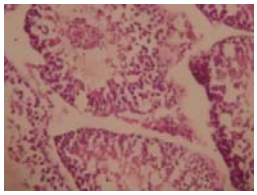
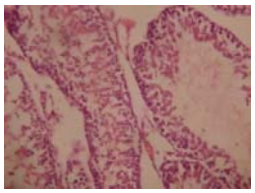
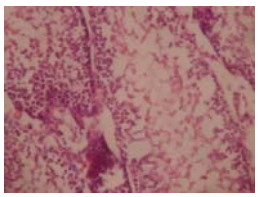
**Pancreas – Figure No: 4.3.9**

**Spleen – Figure No: 4.3.10**

**Stomach – Figure No: 4.3.11**

**Testis – Figure No: 4.3.12**

## 4.4 Pharmacology study

**Results of *Arithiraadhi chooranam***

The acute toxicity study revealed the absence of lethality among the tested animals when the *Arithirathi Chooranam* was administered as a single dose upto 5000 mg/kg). There were no signs of any gross behavioral changes except for mild tremors indicating the safe use of the *Arithirathi Chooranam*. The ability of a hepatoprotective drug to reduce the injurious effects or to preserve the normal hepatic physiological mechanisms, which have been disturbed by a hepatotoxin, is the index of its protective effects. Although serum enzyme levels and ascorbic acid in urine are not a direct measure of hepatic injury, they show the status of the liver. The lowering of enzymes level are definite indication of hepatoprotective action of the drug.

Liver possesses a unique metabolism and plays a pivotal role in the removal of substances from the portal circulation due to which it is susceptible to toxicity of drugs, xenobiotics, and oxidative stress. The two distinct pathways in liver metabolism occur via cytochrome p-450 and GSH-peroxidase. The current treatment for hepatotoxicity includes drugs which influence the p-450 enzyme mechanism either by inhibiting (amiodarone, cimetidine, ciprofloxacin, etc.) or inducing (rifampicin, carbamazepine, phenobarbital, phenytoin) the metabolic activity of enzymes. Necrosis or membrane damage releases the enzymes into circulation and hence it can be measured in the serum.

The reversal of increased serum enzymes in $CCl_4$-induced liver damage by the *Arithirathi Chooranam* may be due to the prevention of the leakage of intracellular enzymes by its membrane stabilizing activity. Amino transferases contribute a group of enzymes that catalyze the interconversion of amino acids and a-keto acids by the transfer of amino groups. These are liver specific enzymes and are considered to be very sensitive and reliable indices for necessary hepatotoxic as well as hepatoprotective or curative effect of various compounds. Both AST and ALT levels increase due to toxic compounds affecting the integrity of the liver cells. Decreased levels of transaminases indicate stabilization of plasma membrane and protection of hepatocytes against damage caused by hepatotoxin.

This is in agreement with the commonly accepted view that serum levels of transaminases return to normal with the healing of hepatic parenchyma and the regeneration of hepatocytes. Alkaline phosphatase is a membrane bound glycoprotein enzyme with a high concentration in sinusoids and endothelium. This enzyme reaches the liver mainly from the bone. It is excreted into the bile; therefore its elevation in serum occurs in hepatobiliary diseases. Serum alkaline phosphatase is related to the functioning of hepatocytes and increase in its activity is due to the increased synthesis in presence of biliary pressure. The results of the present study indicate that the test groups probably stabilize the hepatic plasma membrane from $CCl_4$-induced damage. Reduction of alkaline phosphatase levels with concurrent depletion of raised bilirubin levels suggests the stability of the biliary function during injury with $CCl_4$.

The liver is known to play a significant role in the serum protein synthesis, being the source of plasma albumin and fibrinogen and also the other important components like α and β-globulin. The liver is also concerned with the synthesis of g-globulin. The metabolic biotransformation of amino acid in liver by synthesis, transamination, etc., may be impaired due to the escape of both non-proteins and protein nitrogenous substances from injured cells as mediated by a raise in the serum enzyme levels of ALP, AST and ALT. Normally, AST and ALP are present in high concentration in liver. Due to hepatocyte necrosis or abnormal membrane permeability, these enzymes are released from the cells and their levels in the blood increases. ALT is a sensitive indicator of acute liver damage and elevation of this enzyme in non hepatic diseases is unusual. ALT is more selectively a liver paranchymal enzyme than AST. Bilirubin is one of the most useful clinical clues to the severity of necrosis and its accumulation is a measure of binding, conjugation and excretory capacity of hypatocyte.

Decrease in serum bilirubin after treatment with the *Arithirathi Chooranam* in liver damage induced by $CCL_4$, indicated the effectiveness of the test drug in normal functional status of the liver. The reduction in the total protein is attributed to the initial damage produced and localized in the endoplasmic reticulum which results in the loss of cytochrome P-450 enzymes leading to its functional failure with a decrease in protein synthesis and accumulation of triglycerides leading to fatty liver. Both the

test groups enhanced the synthesis of TP which accelerates the regeneration process and the protection of liver cells. Therefore, the increased level of total protein in serum indicates the hepatoprotective activity.

Inhibition of bile acids synthesis from cholesterol, which is synthesized in liver or derived from plasma lipids leading to increase in cholesterol levels, was also resulted due to $CCl_4$ intoxication. Significant suppression of cholesterol levels by the test groups suggests that the bile acids synthesis inhibition was reversed. The extent of hepatic damage is assessed by histological evaluation along with the level of various biochemical parameters in circulation. The animals in the $CCl_4$ group showed severe hepatotoxicity evidenced by profound steatosis, centrilobular necrosis, ballooning degeneration, nodal formation and fibrosis as compared to the normal hepatic architecture of the normal group animals. Test drug Arithirathi Chooranam showed the healing of damaged parenchyma which was comparable to that of the standard group treated with silymarin.

The efficacy of any hepatoprotective drug is dependent on its capacity of either reducing the harmful effect or restoring the normal hepatic physiology that has been disturbed by a hepatotoxin. The silymarin group and both the test groups decreased $CCl_4$ induced elevated enzyme levels, indicating the protection of structural integrity of hepatocytic cell membrane or regeneration of damaged liver cells. Histopathological examinations of the liver tissues provided supporting evidence for the results obtained in the biochemical analysis.

Liver for rats treated with CCl4 alone showed multiple focal areas of hepatocellular necrosis, degeneration and fatty changes. But in the standard drug treated groups, few areas of necrosis only were observed. In the animals treated with extract at low dose along with CCl4, mild focal hepatocytes necrosis was observed. In the animals treated with extract at medium dose along with CCl4, mild hepatocytes swelling and sinusoidal dilation was observed. But in the animals treated with extract at high dose along with CCl4, Normal central vein and hepatocytes along with few areas of necrosis only were observed.

**Table No: 4.4.1 Effect of Arithirathi Chooranam on serum constituents in $CCl_4$ induced hepatotoxic rats.**

| Group | Treatment | Dose | AST(i.u/l)*** | ALT(i.u/l)*** | ALP(i.u/l)*** | T.P(g/dl)*** | Albumin (g/dl)*** | Bilirubin (mg/dl)*** |
|---|---|---|---|---|---|---|---|---|
| Normal | --- | | 49.50±0.99 | 7.33±0.33 | 69.33±0.49 | 5.23±0.02 | 2.28±0.54 | 0.25±0.02 |
| Control | $CCl_4$ | (3ml/kg) | 63.17±0.48 | 13.0±0.73 | 86.33±0.97 | 5.77±0.05 | 4.33±0.03 | 0.80±0.02 |
| Test I | $CCl_4$+*Arithirathi Chooranam* | 250mg/kg | 55.0±0.82 | 8.50±0.21 | 78.83±0.79 | 5.40±0.04 | 2.68±0.05 | 0.45±0.04 |
| Test II | $CCl_4$+*Arithirathi Chooranam* | 500mg/kg | 53.83±0.48 | 8.0 ± 0.37 | 76.50±0.73 | 5.30±0.04 | 2.58±0.05 | 0.42±0.03 |
| Standard | $CCl_4$+Silymarin | 50mg/kg | 50.17±0.70 | 7.67±0.21 | 70.33±0.76 | 5.25±0.03 | 2.33±0.03 | 0.28±0.03 |

Values are as mean ± SEM (n=6)

Values are statistically significant at ***P<0.001

Comparison made between Group II Vs Group I

Group III, IV, V Vs Group II.

**Table No:4.4.2 Effect of Arithirathi Chooranam on hematological Parameters in $CCl_4$ induced hepatotoxic rats.**

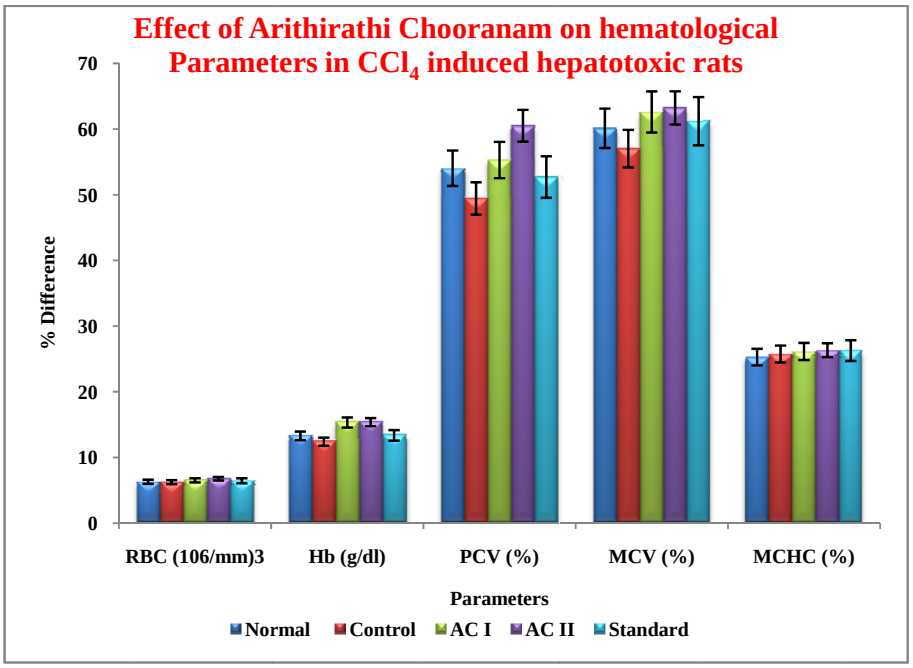
| Group | Treatment | Dose | RBC $(10^6/mm)^3$ | Hb (g/dl) | PCV (%) | MCV (%) | MCHC (%) |
|---|---|---|---|---|---|---|---|
| Normal | --- | | 6.30±0.32 | 13.29±0.45 | 54.05±2.23 | 60.13±1.44 | 25.29±0.17 |
| Control | $CCl_4$ | (3ml/kg) | 6.24±0.25 | 12.40±0.28 | 49.44±2.20 | 57.04±0.60 | 25.76±0.22 |
| Test I | $CCl_4$+*Arithirathi Chooranam* | 250mg/kg | 6.52±0.23 | 15.33±0.33*** | 55.30±2.13 | 62.62±0.52** | 26.15±0.40 |
| Test II | $CCl_4$+*Arithirathi Chooranam* | 500mg/kg | 6.76±0.34 | 15.38±0.30*** | 60.52±2.18* | 63.24±0.36*** | 26.34±0.21 |
| Standard | $CCl_4$+Silymarin | 50mg/kg | 6.45±0.28 | 13.36±0.31 | 52.71±2.31 | 61.22±0.62** | 26.28±0.40 |

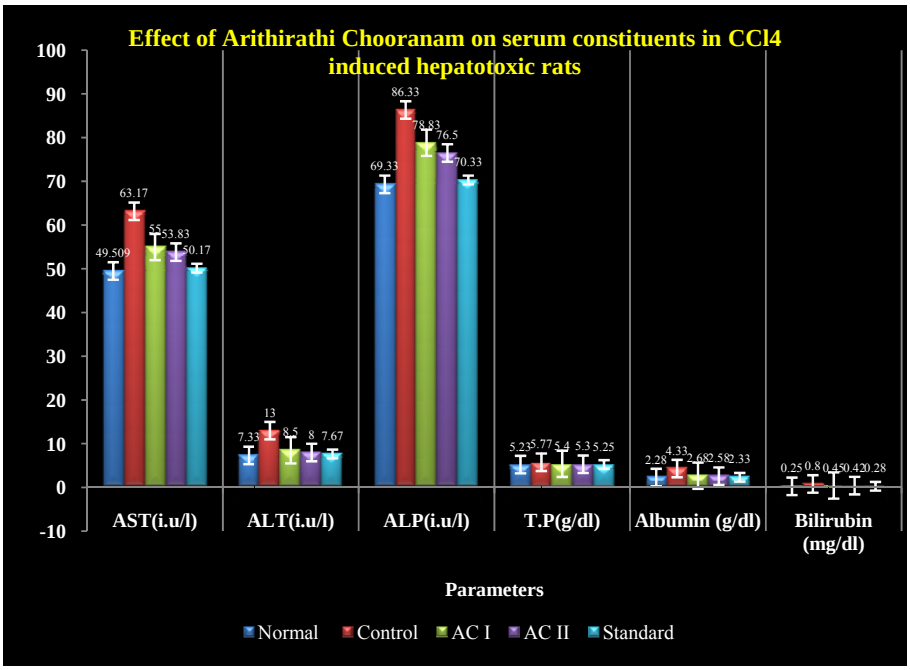
Values are as mean ± SEM (n=6)

Values are statistically significant at *P<0.05, **P<0.01, ***P<0.001

Comparison made between Group II Vs Group I

Group III, IV, V Vs Group II.


Effect of Arithirathi Chooranam on hematological Parameters in $CCl_4$ induced hepatotoxic rats
% Difference
70
60
50
40
30
20
10
0
RBC (106/mm)3
Hb (g/dl)
PCV (%)
MCV (%)
MCHC (%)
Parameters
Normal
Control
AC I
AC II
Standard

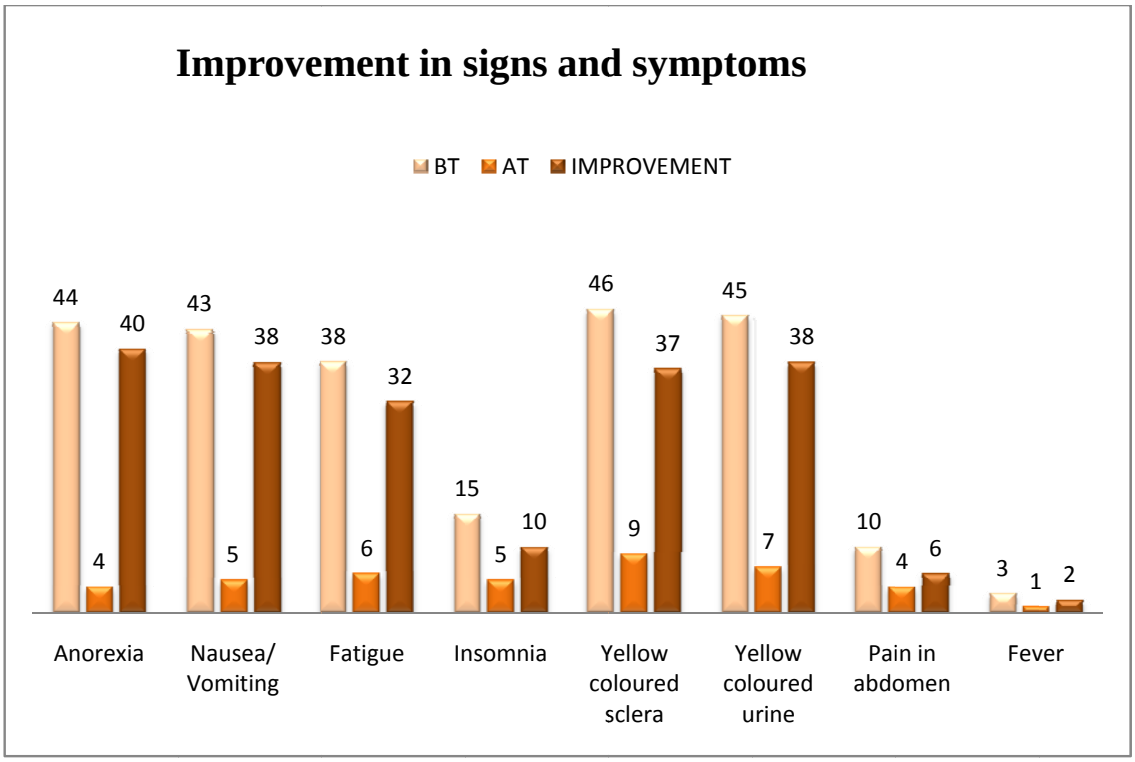

Effect of Arithirathi Chooranam on serum constituents in CCl4 induced hepatotoxic rats
100
90
80
70
60
50
40
30
20
10
0
-10
49.509
63.17
55
53.83
50.17
7.33
13
8.5
8
7.67
69.33
86.33
78.83
76.5
70.33
5.23
5.77
5.4
5.3
5.25
2.28
4.33
2.68
2.58
2.33
0.25
0.8
0.45
0.42
0.28
AST(i.u/l)
ALT(i.u/l)
ALP(i.u/l)
T.P(g/dl)
Albumin (g/dl)
Bilirubin (mg/dl)
Parameters
Normal
Control
AC I
AC II
Standard

## CLINICAL ASSESSMENT

### Improvement in signs and symptoms

**Table No: 4.5.24**

| SL. NO | SIGNS AND SYMPTOMS | NO. OF PATIENTS | | | |
|---|---|---|---|---|---|
| | | BT | AT | IMP. | IMPROVEMENT (%) |
| 1 | Anorexia | 44 | 4 | 40 | 91 |
| 2 | Nausea/ Vomiting | 43 | 5 | 38 | 88 |
| 3 | Fatigue | 38 | 6 | 32 | 84 |
| 4 | Insomnia | 15 | 5 | 10 | 67 |
| 5 | Yellow coloured sclera | 46 | 9 | 37 | 80 |
| 6 | Yellow coloured urine | 45 | 7 | 38 | 84 |
| 7 | Pain in abdomen | 10 | 4 | 6 | 60 |
| 8 | Fever | 3 | 1 | 2 | 67 |



**Inference:**

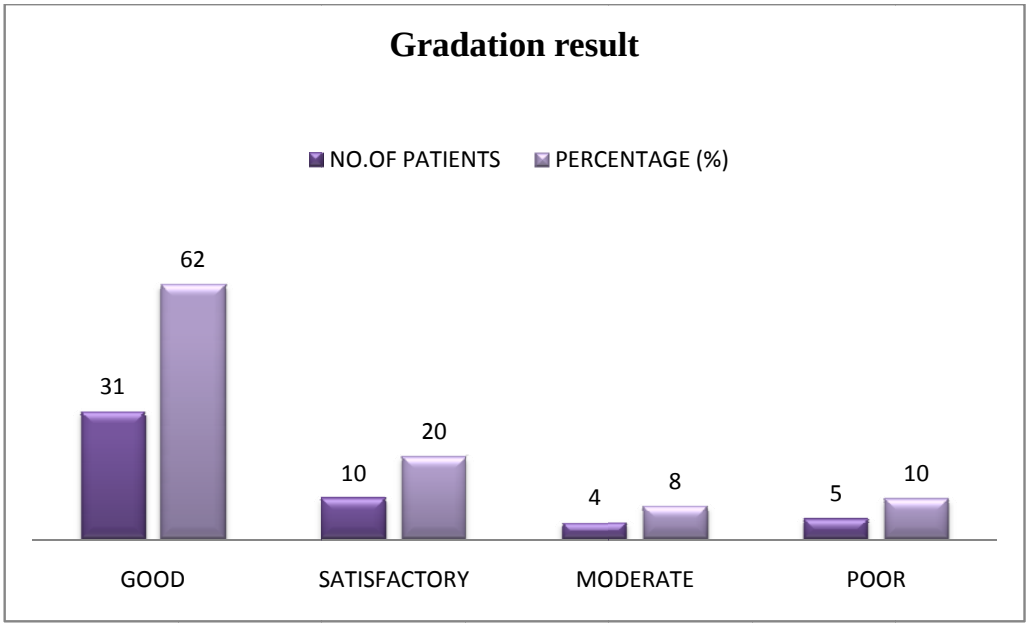
Among 50 patients,

- 40 out of 44 patients were relieved from Anorexia.
- 38 out of 43 patients were relieved from Nausea/ Vomiting.
- 32 out of 38 patients were relieved from Fatigue.
- 10 out of 15 patients were relieved from Insomnia.
- 37 out of 46 patients were relieved from Yellow coloured eyes.
- 38 out of 45 patients were relieved from Yellow coloured urine.
- 6 out of 10 patients were relieved from pain in abdomen.
- 2 out of 3 patients were relieved from Fever.

**Table No: 4.5.25**

**Gradation result**

| SL. NO | LEVEL OF IMPROVEMENT | NO.OF PATIENTS | PERCENTAGE (%) |
|---|---|---|---|
| 1 | Good | 32 | 64 |
| 2 | Satisfactory | 10 | 20 |
| 3 | Moderate | 5 | 10 |
| 4 | Poor | 6 | 12 |
| TOTAL | | 47 | 100 |



## STATISTICAL ANALYSIS

**Descriptive statistical for improvement Of liver function test In *kamalai***

**Paired *t* test results:**

**AST in Jaundice Patients:**

**P value and statistical significance:**

The two-tailed P value is less than 0.0001

By conventional criteria, this difference is considered to be extremely statistically significant.

**Confidence interval:**

The mean of Group One minus Group Two equals 67.359

95% confidence interval of this difference: From 53.440 to 81.278

**Intermediate values used in calculations:**

t = 9.7969

df = 38

standard error of difference = 6.876

**Table No: 4.5.26**

| Treatment | No of Patients | Mean | S.D | S.E.M | N |
|---|---|---|---|---|---|
| Before treatment | 40 | 113.920 | 52.024 | 8.226 | 40 |
| After treatment | 40 | 46.282 | 13.533 | 2.167 | 39 |

**ALT (Alanine transaminase):**

The elevated ALT (Alanine transaminase) /serum glutamic pyruvic transaminase (SGPT) levels were also reduced significantly, when compared to the pre-treatment values, the two-tailed P value is less than 0.0001.By conventional criteria, and this difference is considered to be extremely statistically significant.

**P value and statistical significance:**

The two-tailed P value is less than 0.0001

By conventional criteria, this difference is considered to be extremely statistically significant.

**Confidence interval:**

The mean of Group One minus Group Two equals 52.077

95% confidence interval of this difference: From 40.564 to 63.590

**Intermediate values used in calculations:**

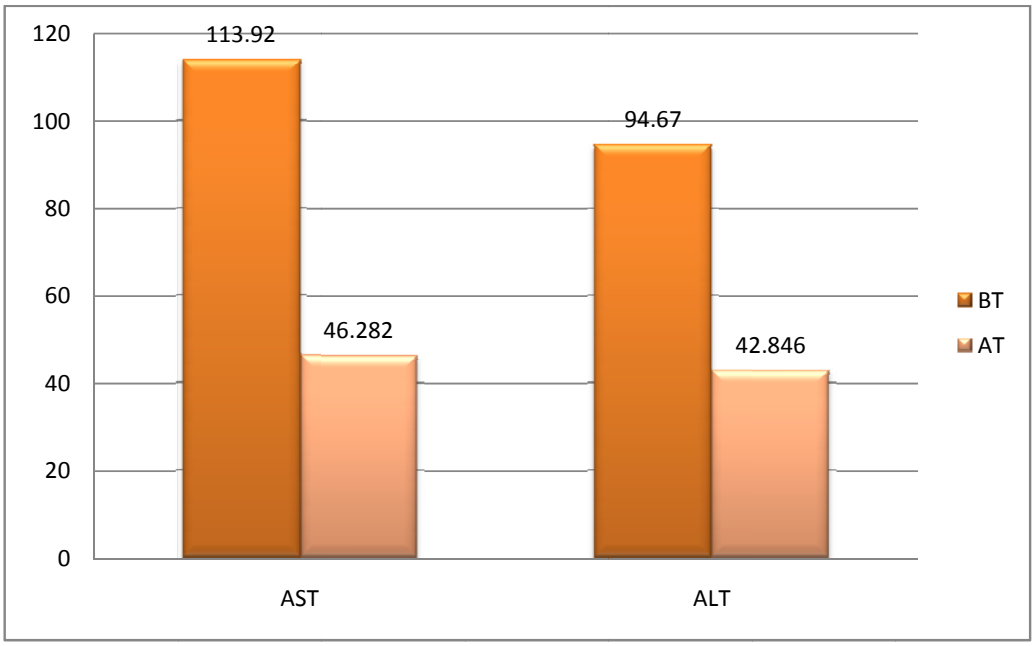
t = 9.1572

df = 38

standard error of difference = 5.687

**Table No: 4.5.27**

| Treatment | No of Patients | Mean | S.D | S.E.M | N |
|---|---|---|---|---|---|
| Before treatment | 40 | 94.670 | 43.975 | 6.953 | 40 |
| After treatment | 40 | 42.846 | 12.881 | 2.063 | 39 |




**ALP: Alkaline Phosphatase:**

The elevated alkaline phosphatase levels were also reduced significantly, when compared to the pre-treatment values, the two-tailed P value is less than 0.0001. By conventional criteria, this difference is considered to be extremely statistically significant.

**P value and statistical significance:**

The two-tailed P value is less than 0.0001

By conventional criteria, this difference is considered to be extremely statistically significant.

**Confidence interval:**

The mean of Group One minus Group Two equals 13.777

95% confidence interval of this difference: From 9.463 to 18.091

**Intermediate values used in calculations:**

t = 6.4652

df = 38

standard error of difference = 2.131

**Table No:4.5.28**

| Treatment | No of Patients | Mean | S.D | S.E.M | N |
|---|---|---|---|---|---|
| Before treatment | 40 | 109.747 | 22.250 | 3.518 | 40 |
| After treatment | 40 | 96.682 | 16.886 | 2.704 | 39 |

**Bilirubin:**

The mean serum bilirubin values were elevated at the time of the enrolment, in all patients in these studies. Cumulative data analysis showed a significant reduction in the mean serum bilirubin level,The two-tailed P value is less than 0.0001By conventional criteria, this difference is considered to be extremely statistically significant.

**P value and statistical significance:**

The two-tailed P value is less than 0.0001

By conventional criteria, this difference is considered to be extremely statistically significant.

**Confidence interval:**

The mean of Group One minus Group Two equals 1.0872

95% confidence interval of this difference: From 0.9143 to 1.2600

**Intermediate values used in calculations:**

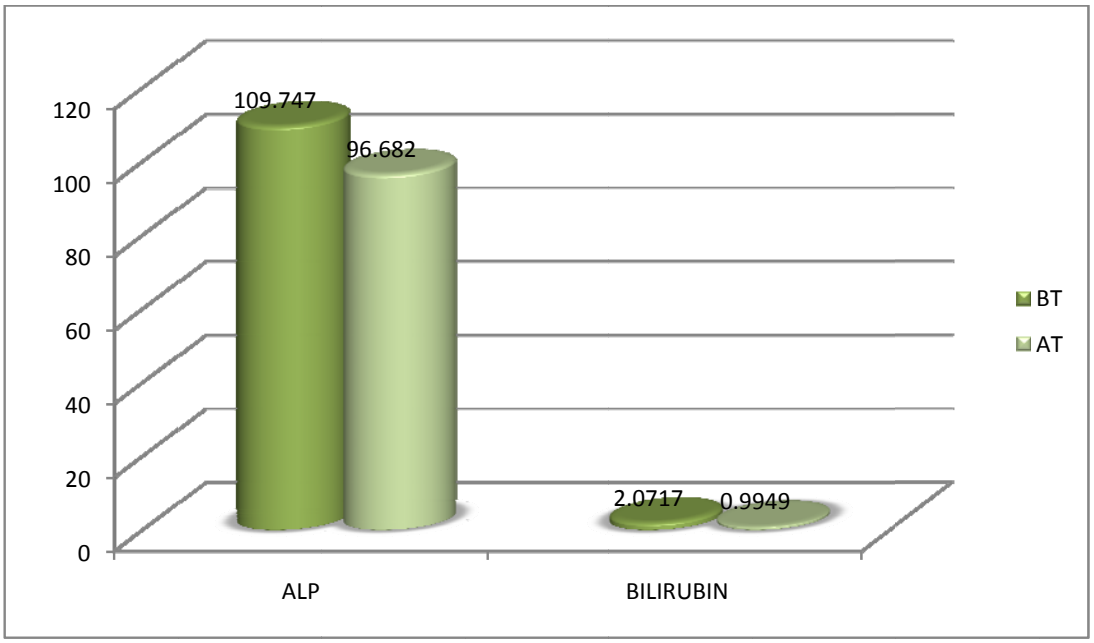
t = 12.7333

df = 38

standard error of difference = 0.085

**Table No: 4.5.29**

| Treatment | No of Patients | Mean | S.D | S.E.M | N |
|---|---|---|---|---|---|
| Before treatment | 40 | 2.0717 | 0.6136 | 0.0970 | 40 |
| After treatment | 40 | 0.9949 | 0.1905 | 0.0305 | 39 |



# CONCLUSION

The test drug *Arithiraadhi chooranam* was selected from the text "*Sarabenthira vaithiya rathnavali"* for the evaluation of safety, efficacy and therapeutic potential in Jaundice.

All the raw drugs for the preparation of test drug were purchased from Tampcol raw drug store and authenticated, then subjected to preparation.

Physico chemical anatysis, Chemical analysis, was showed that *Curcuma longa, Picrorhiza kurao,* and *Terminalia chebula* having phytochemicals which are hepatoprotecting agents.

Tannins and flavanoids of *Phyllanthus emblica* contain anti oxidant and hepatoprotective properties.

From the acute toxicity study as per OECD guideline 425, it was concluded that the test drug *Arithiraadhi chooranam* is a safety drug.

*Arithiraadhi chooranam* did not produce any oral acute or sub acute toxicity in both female and male rats. Overall results suggest that *Arithiraadhi chooranam* is relatively safe in rats.

The finding of the pre clinical study suggests that effective role of *Arithiraadhi chooranam* on jaundice.

# SUMMARY

The herbo mineral drug *Arithiraadhi chooranam* was prepared as per siddha literature. This drug was subjected to various studies by the author.

*Arithiraadhi chooranam* was selected for this study to establish the safety and efficacy of its Hepatoprotective activity on *Kamalai.*

To collect the information about the drug, various text books, Literature were referred. From them, the author came to an idea about the drug and its efficacy on *Kamalai.*

Acute and Sub acute toxicological studies show strong evidence of the nontoxic effect of the *Arithiraadhi chooranam.*

The pharmacological analysis showed that the drug has got significant Hepatoprotective Efficacy.

In clinical study, the drug has showed improvement in 62% of cases.

The patients were responding well from the beginning of the treatment and no adverse effects were reported.

This present study suggests that *Arithiraadhi chooranam* has remarkable medicinal value against the disease *Kamalai.*

# BIBILIOGRAPHY


1. Agaththiyar gunavagadam
2. Agaththiyar paripooranam -1200
3. Agaththiyar paripooranam-400
4. Anuboga vaithiya Navaneetham. Part 1 to 7
5. Aviyalikum amutha surakkam
6. Compendium of Indian Medicinal Plants
7. Danvadri nigandu
8. Davidson's Principles and Practice of Medicine.
9. Fundamental of Bio-Chemistry for Medical student.
10. Gunapadam Mooligai Vaguppu ( Murugesa Mudhaliyar )– Indian Medicine and homeopathy Dept. – Chennai-106.
11. Gunapadam Thathu – Seeva Vaguppu (Part (2 & 3) Dr.R .Thiyagarajan. L.I.M. Indian Medicine and Homeopathy Dept. Chennai-106.
12. Indian Material Medica Dr. K.M. Nadkarni
13. Kannusamy Parambarai Vaidhiyam
14. Manual of Practical Medicine. . Dr. R. Alagappan.
15. Marunthu sei iyalum kalaium
16. Medical Physiology. Prof. Guyton. MD.
17. Noi nadal noi muthal nadal Part 1
18. Noi nadal noi muthal nadal Part 2

19. Papiya Mitra Mazumder*, D Sasmal and R Arivudai Antiulcerogenic and antioxidant effects of Coccinia Linn.) Voigt leaves on aspirin-induced gastric ulcer in rats Natural Product Radiance, Vol. 7(1), 2008,

20. Pathartha guna villakam

21. Patharththa guna sinthamani

22. Pathinen siddharkal nadi sasthiram

23. Pathologic basic of Disease. Dr. Robins.

24. Sarebenthirar gunma noi sigichai

25. Shanmugavelan, Siddha Science of Longevity and Kalpa Medicine; Published by Directorate of Indian medicine,1992.

26. Siddha Maruthuvam – Fr. Kuppusami Mudhaliar

27. Siddha Maruthuvanga Churukkam, Dr. K.S. Uthamarayan.

28. Siddha vathya thirattu

29. Sikicha rathana Deepam

30. T.V. Sambasivampillai agarathi Inidan medicine and Homeopathy Durai.

31. Text Book of Bio-Chemistry by SathyaNarayanah

32. Text Book of Medicine. Prof. K.V. Krishnadas.

33. Text Book of Medicine. Prof. P.C . Das.

34. Thanvanthiri nigandu

35. The Wealth of India

36. Theraiyar venba

37. Theriyar kaapiyyam

38. Theriyar venba.

39. Tripathi K.D., Gastrointestinal Drugs: Drugs for peptic ulcers. In: Essentials of Medical Pharmacology. (Jaypee Brothers Medical Publishers (P) Ltd. New Delhi, 1999) 628-642.

40. Udal thathuvam

41. Indian plants and drugs with their medical properties and uses

42. Theraiyar maga karisal

43. Sarabendhira vaithiya rathnaavali

44. Sarabendhira vaithiya muraigal

45. Pararaasa sekaram

46. Yugi sinthamani

47. Aavi alikum amudha murai surukam

48. Saraku suthi sei muraigal

49. Noikaluku siddha parikaaram

50. Oxford text book of medicine Vol.1

51. Price's text book of practice of medicine 12th edition

52. The clinician's guide to Acid peptic disorders and motility disorders of GI tract

53. Kokate Text book of Pharmacognosy

54. Pharmacology and pharmacotherapeutics - Satoskar

55. Easu, K. 1964. Plant Anatomy John Wiley and sons. New York. Pp.767. Easu, K. 1979. Anatomy of seed Plants. John Wiley and sons. New York.

Pp. 550.

56. Gamble, J.S 1935. Flora of the Presidency of Madras. Vol. I, II, & III. Botanical

Survey of India, Calcutta, India.

57. Henry, A.N; Kumari, G.R. and Chitra, V. 1987. Flora of Tamilnadu, India. Vol.3

58. Botanical Survey of India, Southern Circle, Coimbatore, India. pp-258.

59. Johansen, D.A. 1940. Plant Microtechnique. Mc Graw Hill Book Co; New York. Pp.523.

60. Mathew, K.M. 1983. The Flora of Tamil Nadu Karnatic Vol.I. Polypetalae.pp.688.

61. . Gamopetalae & Monochlamydae Vol.3 pp.689-1540. The Ranipat Herbarium, St.John's College, Tiruchirappalli, India.

62. Metcalfe, C.R. and Chalk, L. 1950. Anatomy of the Dicotyledons. Vol. I&II. Clarendon Press, Oxford.

63. Metcalfe, C.R. and Chalk, L. 1979. Anatomy of the Dicotyledons. Vo1.I. Clarendon Press, Oxford.pp.276.

64. O'Brien, T.P; Feder, N. and Mc Cull, M.E. 1964. Polychromatic Staining of Plant Cell walls by toluidine blue-O.Protoplasma; 59:364-373.

65. Sass, J.E. 1940. Elements of Botanical Microtechnique. McGraw Hill Book Co; New York. pp.222.

66. Wallis, T.E.1985. Text Book of Pharmacognosy, CBS Publishers and Distributors, Shahdara, Delhi, India.

67. YogaNarasimhan, S.N.2000.Medicinal Plants of India. Vo1.II.Tamailnadu. Regional Research Institute (Ay.) Bangalore, India.p.715

68. Ajay KG, Neelam M (2006), Hepatoprotective activity of aqueous and ethanolic extract of Chamomile capitula in paracetamol intoxicated albino rats. American J Pharmacol Toxicol, 1(1), pp 1720.
69. Anand KK, Gupta VN, Rangari VD, Chandan BK. Structure and hepatoprotective activity of bioflavonoid from Canarium manii. Planta Med 1992;58:493-5.
70. Arulkumaran KS, Rajasekaran A, Ramasamy A, Jegadeesan M, Kavimani S, Somasundaram A (2009), Cassia roxburghii seeds protect liver against toxic effects
71. Ashok S. K., Somayaji S.N. and BairyK.L. Hepatoprotective effects of Ginkgo biloba against carbon tetrachloride induced hepatic injury in rats. Indian J Pharmacol 33(2): 260-6 (2001).
72. Bahar A, Tanveer A, Shah AK. Hepatoprotective activity of Luffa echinata fruits. J Ethnopharmacol 2001;76:187-9.
73. Bardhan P, Sharma SK, Garg NK (1985), in vitro effect of an ayurvedic liver remedy on hepatic enzymes in carbon tetrachloride treated rats. Indian Journal of Medical Research, 82, pp 359364.
74. Bishayee, A., Sarkar, A., Chatterjee, M, The hepatoprotective activity of Carrot (Daucus carota L) against carbon tetrachloride intoxication in mouse liver. J. Ethnopharmacol. 1995, 47, 69–74.
75. Boyd EH and Bereczky GM. Liver necrosis from paracetamol. Br J Pharmacol. 1966; 26: 606-614.
76. Deb AC. Liver function tests. Fundamentals of biochemistry. 8th edition; Kolkata: 2008:581:6.

77. Gupta AK and Misra N. Hepatoprotective Activity of Aqueous Ethanolic Extract of Chamomile capitula in Paracetamol Intoxicated Albino Rats. American Journal of Pharmacology and Toxicology. 2006; 1: 17-20.
78. Gupta, A.K. and N. Misra (2006), Hepatoprotective Activity of Aqueous Ethanolic Extract of Chamomile capitula in paracetamol intoxicated albino rats. A m .J. Pharm. Toxicol., 1, pp 17 20.
79. Harshmohan. The liver, biliary tract, exocrine and pancreas: Textbook of Pathology. 4th ed. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2002:22-4,569-80.
80. Hinson. J.A. Pohl. Monks, Gillete JR. Acetaminophen induced hepatotoxicity. Life Sci 1981; 29: 107-116.
81. HuiMei L, HsienChun T, ChauJong W, JinJin L, ChiaWen L, FenPi C (2008), Hepatoprotective effects of Solanum nigrum Linn extract against CCl4iduced oxidative damage in rats. ChemicoBiological Interactions, 171, pp 283–293
82. Irwin. The organization of screening. In: Robert A Turner. Screening methods in Pharmacology. India: Elsevier; 2009. p. 22-40.
83. Jalalpure SS, Patil MB. Hepatoprotective activity of the fruits of *Piper longum*. Indian J Pharm Sci. 2003;65:363.
84. Karan, M., Vasisht, K., Handa, S.S. Antihepatotoxic activity of Swertia chirata on carbon tetrachloride induced hepatotoxicity in rats. Phytotherapy Research. 1999. 13, 24–30.
85. Krishna V, Mankani KL, Shanthamma C. Evaluation of hepatoprotective activity of stem bark of Diospyros cordifolia. Indian J Pharm Sci. 2005;67:106.

86. Kumar G, Sharmila Banu G, Vanitha Pappa P, Sundararajan M, Rajasekara Pandian M. Hepatoprotective activity of Trianthema portulacastrum L. against paracetamol and thioacetamide intoxication in albino rats. J Ethnopharmacol 2004;92:37-40.
87. Kumar SR, Mishra SH. Hepatoprotective activity of the whole plants of Fumaria indica. Indian J Pharm Sci. 1997;59:165.
88. Lin, C.C., Yen, M.H., Lo, T.S., Lin, J.M., Evaluation of the hepatoprotective and antioxidant activity of Boehmeria nivea var. nivea and B. nivea var. tenacissima. J. Ethnopharmacol. 1998, 60, 9–17.
89. Marshal M, Kalpan MD. Understanding liver function tests 2007. Obtained from world wide web: http://ocw.tufts.edu/data/48/593572. (Cited on12th July 2011).
90. Nkosi CZ, Opoku AR and Terblanche SE. Effect of pumpkin seed (Cucurbita pepo) protein isolate on the activity levels of certain plasma enzymes in CCl4-induced liver injury in lowprotein fed rats. Phy.the.Res.2005; 19: 341–345.
91. Prescott, L.F. Wringht. N. Roscoe. P and Brown, S.S. Plasma Paracetomol half-life and hepatic necrosis in patients with paracetomol overdose Lancet. I; 519-522.
92. Rane A, Grampurohit ND. Hepatoprotective activity of Petrocarpus marsupium and Butea frondosa koen. Indian J Pharm Sci. 1998;65:182.
93. Reitman S and Frankel S. In vitro determination of tranaminase activity in serum. Am. J. Clin. Pathol. 1957; 28: 56.
94. Richard OR. A new direction in the study of carbon tetrachloride hepatotoxicity. Life Sci 1983;33:401-8.
95. Roger walker, Cate W. Liver diseases. Clinical pharmacy and therapeutics. Churchil living stone: 4th edtion; London: 2007:221-2.

96. Sanmugopriya, E., Venkataraman,S., Studies on hepatoprotective and antioxidant actions of Strychnos potatorum Linn. seeds on CCl4-induced acute hepatic injury in experimental rats J. Ethnopharmacol., 2006. (105). 154-160.
97. Seakins A, Robinson DS. The effect of the administration of carbon tetrachloride on the formation of plasma lipoproteins in rats. Biochem J 1963;86:401-7.
98. Sharma A. Chakraborti. K.K., Handa S.S. Anti hepatotoxic activity of some Indian herbal formulation as compared to silymarin Fitoterapia. 1991; 62:299-35.
99. Suja, S.R., Latha, P.G., Pushpangadan, P., Rajasekharan, S., Evaluation of hepatoprotective effects of Helminthostachys Zeylanica (L.) Hook against carbon tetrachloride induced liver damage in Wistar rats. J. Ethnopharmacol.2004, 92, 61–66.
100. Suresh Kumar SV, Sujatha C, Syamala J, Nagasudha B, Mishra SH. Hepatoprotective activity of extracts from Pergularia daemia Forsk. against Carbon tetrachloride induced toxicity in rats. Phcog Mag 2007;3:11.
101. Tran QI, Adnyana IK, Tezuka Y, Nagaoka T, Tran QK, Kadota S. Triterpene saponins from Vietnamese ginseng (Panax vietnamensis) and their hepatocyte protective activity. J Nat Prod 2001;64:456-61.
102. Shah M et al. Evaluation ofthe effect of aqueous extract from powders of root, stem, leaves and whole plant of phyllanthus debilis against CCL4 induced rat liver dysfunction. Indian Drugs. 2002; 39: 333-337.
103. Ariyphisi I., Toshiharu A., Sugimura F., Abe M., Matsuo Y. and Honda T. Recurrence during maintenance therapy with histamine H2 receptors antagonist in cases of gastric ulcers. Nikon University Journal Medical 28 :69-74 (1986).

104. Basil MD, Howard MS. Clinical gastroenterology. In: Companion Handbook. 4th ed. USA: McGraw-Hill; 1995.

105. Chan P.K. and Hayes A.W. (1994). Chap. 16. Acute Toxicity and Eye Irritancy. *Principles and Methods of Toxicology*. Third Edition. A.W. Hayes, Editor. Raven Press, Ltd., New York, USA.

106. Falk G.W. Disease of the stomach and duodenum. In: Andreoli ThE, editor. Cecil essentials of medicine. 5th ed. Edinburgh: W.B. Saunders Company; 334-343 (2001).

107. Hogan, D. L.; Ainsworth, M. A. and Ibensberg, J. I. (1994), Gastro duodenal bicarbonate secretion eliminate. Pharmacol. and Ther., 8, 475-479.

108. Lipnick R.L., Cotruvo J.A., Hill R.N., Bruce R.D., Stitzel K.A., Walker A.P., Chu I., Goddard M., Segal L., Springer J.A., and Myers R.C. (1995). Comparison of the Up-and-Down, Conventional $LD_{50}$ and Fixed Dose Acute Toxicity Procedures. Fd. Chem. Toxicol., 33, 223-231.

109. Nash J, Lynn L, Deakin M. Histamine $H_2$-receptor antagonist in peptic ulcer disease. Evidence for a prophylactic use. Drugs 1994;47:862-71.

110. OECD Guidelines For The Testing Of Chemicals For Acute Oral Toxicity 425– Up-And-Down-Procedure Adopted On 3 October 2008.

111. Robert, A. (1979), Cytoprotection by prostaglandins. Gastroenter, 77, 761-762.

112. S.K. Kulkarni, Handbook of experimental Pharmacology, (Vallabh Prakashan, New Delhi, 2002) pp148.

113. Szabo, S. (1987), Mechanisms of mucosal injury in the stomach and duodenum: Time-sequence analysis of morphologic, functional, biochemical and histochemical studies. Scan. J. Gastroent, 22: (127), 21-28.

114. Tripathi K.D., Gastrointestinal Drugs: Drugs for peptic ulcers. In: Essentials of Medical Pharmacology. (Jaypee Brothers Medical Publishers (P) Ltd. New Delhi, 1999) 628-642.

115. Vane, J. R. (1971), Inhibition of prostaglandin synthesis as a mechanism of action for aspirin-like drugs. Nature (New Biol.), 231, 232-235.

**Form: I**

## CONSENT FORM

I certify that I have disclosed all the details about the study in the terms readily understood by the patient.

**DATE:** **SIGNATURE**

**NAME**

## CONSENT BY THE PATIENT

I have been informed to my satisfaction by the attending physician the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigations to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trial at any time during the course of the trial without having to give reasons for doing so.

I ,exercising my free power of choice , hereby give my consent to be included as a subject in the clinical trial of **…….**for the treatment of**……..**

**DATE:** **SIGNATURE**

**NAME**

## ஒப்புதல் படிவம்

ஆய்வுகுறித்தஅத்தனைதகவல்களையும் நோயாளிஎளிதில் புரிந்துகொள்ளும் வகையில் நோயாளிக்குவிளக்கியுள்ளேன் என்றுஉறுதியளிக்கிறேன்

தேதி: ஆய்வாளரின் கையொப்பம்:

பெயர்:

## நோயாளியின் ஒப்புதல்

இந்தஆய்வுகுறித்த முழு தகவல்கள்,மருந்தின் தன்மை,எனதுஉடல் நலன் குறித்தஆய்வுகள்,ஆய்வுக்கானமருத்துவபரிசோதனைகள் மற்றும் சிகிச்சைவிபரங்கள் ஆகியஅனைத்தும் மருத்துவரால் முழுமையாகவிளக்கிக் கூறப்பட்டுள்ளது.

இந்தஆய்விலிருந்துஎந்தநிலையிலும்,எவ்விதகாரணமுமின்றிவிலகிக்கொள்ளஎனக்கு முழு சுதந்திரம் உள்ளதுஎன்பதையும் அறிந்திருக்கிறேன்.

இந்தஆய்வில்,.....ஒருபயனாளியாகஎன்னைஉட்படுத்திக் கொள்ளஏவ்விதமானநிர்பந்தமுமின்றிமுழுமனதுடன் சம்மதிக்கிறேன் என்பதைத் தெரிவித்துக் கொள்கிறேன்.

தேதி: கையொப்பம்:

பெயர்:

**1. DEPARTMENT OF POST GRATUATE GUNAPADAM BRANCH,**

**GOVERNMENT SIDDHA MEDICAL COLLEGE,**

**CHENNAI - 106.**

**ARIGNAR ANNA GOVERNMENT HOSPITAL FOR**

**INDIAN MEDICINE AND HOMEOPATHY, ARUMBAKKAM,**

**CHENNAI - 106.**

**OPEN CLINICAL TRAIL PHASE II B**

**ANTI ULCER ACTIVITY OF**

***"PANAI POO CHOORANAM"***

**FORM II**

1. Centre :
2. Code no : Level of study: OPD/IPD
3. Name of the patient :
4. Address :

5. Age : : sex: Male ☐ Female ☐
6. Educational Status :
7. Occupation :
8. Income :
9. Religion : H ☐ M ☐ CH ☐ S ☐
10. Marital Status :

11. Date of Admission :

12. Date of Discharge :

13Diagnosis :

**PERSONAL HISTORY:**

11. Food habits : Veg ☐ Non Veg ☐ Veg/Egg ☐
12. Addiction : None ☐ Smoking ☐ Snuff ☐
    Ganja ☐ Alcohol ☐ Opium ☐
13. Sleep : Good ☐ Distributed ☐ Insomnia ☐
14. Presence of anxiety : Yes ☐ No ☐
15. Naadi : Vatham ☐ Pitham ☐ Kapam ☐ Thontham ☐

**FAMILY HISTORY:**

1. Hypertension : Yes ☐ NO ☐
2. Diabetes mellitus : Yes ☐ NO ☐
3. Tuberculosis : Yes ☐ NO ☐
4. IHD/MI/MS/AS : Yes ☐ NO ☐
5. If any other disease specify : Yes ☐ NO ☐

**PRESENTING SYMPTOMS:**

| | | | Yes | No | Duration (WEEKS) |
|---|---|---|---|---|---|
| 1. | Pain | : | ☐ | ☐ | _____ |
| 2. | Vomiting | : | ☐ | ☐ | _____ |
| 3. | Indigestion | : | ☐ | ☐ | _____ |
| 4. | Sore tongue | : | ☐ | ☐ | _____ |
| 5. | Diarrhoea | : | ☐ | ☐ | _____ |
| 6. | Constipation | : | ☐ | ☐ | _____ |
| 7. | Haematemesis | : | ☐ | ☐ | _____ |
| 8. | Loss of appetite | : | ☐ | ☐ | _____ |
| 9. | Thirst | : | ☐ | ☐ | _____ |
| 10. | Dysphasia | : | ☐ | ☐ | _____ |
| 11. | Flatulence | : | ☐ | ☐ | _____ |
| 12. | Heart-burn | : | ☐ | ☐ | _____ |
| 13. | If any other disease | : | _____ | | |

**HISTORY OF PRESENT ILLNESS:**

| | | |
|---|---|---|
| 1. | Pain | : |
| 2. | Vomiting | : |
| 3. | Indigestion | : |
| 4. | Sore tongue | : |
| 5. | Diarrhoea | : |
| 6. | Constipation | : |
| 7. | Haematemesis | : |
| 8. | Loss of appetite | : |
| 9. | Thirst | : |
| 10. | Dysphasia | : |
| 11. | Flatulence | : |
| 12. | Heart-burn | : |
| 13. | Bloating & abdominal fullness | : |
| 14. | Water brash | : |
| 15. | Nausea and copious vomiting: | |
| 16. | Weight loss | : |
| 17. | Melena | : |
| 18. | If any other disease | |

**PAST HISTORY:**

| | | | | | | |
|---|---|---|---|---|---|---|
| 1. | Date of diagnosis | : _____ | | | | |
| 2. | Duration of disease | : _____ | | | | months/years |
| 3. | Source of any infection | : _____ | | | | |
| 4. | Gastrointestinal bleeding | : Yes | ☐ | No | ☐ | If Yes, when? _____ |
| 5. | Cancer(GI tract) | : Yes | ☐ | No | ☐ | |
| 6. | Cancer(other organs) | : Yes | ☐ | No | ☐ | |
| 7. | Sedentary life style | : Yes | ☐ | No | ☐ | |
| 8. | Achlorhydria | : Yes | ☐ | No | ☐ | |
| 9. | Tumours in stomach | : Yes | ☐ | No | ☐ | |
| 10. | Tuberculosis (GI tract) | : Yes | ☐ | No | ☐ | |
| 11. | Diabetes | : Yes | ☐ | No | ☐ | |
| 12. | Nephritis | : Yes | ☐ | No | ☐ | |
| 13. | Syphilis | : Yes | ☐ | No | ☐ | |

14. AIDS : Yes ☐ No ☐
15. Pt.underwent any surgery : Yes ☐ No ☐
16. UTI : Yes ☐ No ☐
17. Others : If Yes , Specify _____

**HISTORY OF PREVIOUS TREATMENT:** Yes No ☐ ☐

**If yes, give details as follows:**

**PHYSICAL EXAMINATION:**

1. Height (cm) :_____
2. Weight (cm) :_____
3. Pulse :_____
4. Blood pressure :_____
5. Temperature :_____
6. RR :_____ /minute
7. Anaemia : Present ☐ Absent ☐
8. Lymphadenopathy : Present ☐ Absent ☐
9. Pigmentation : Present ☐ Absent ☐

**PRESENTING SIGNS:**

1. Bloating and abdominal fullness : Present ☐ Absent ☐
2. Water brash (Rush of saliva after an episode of Regurgitation to Dilute the Acid in oesophagus) : Present ☐ Absent ☐
3. Nausea and copious vomiting : Present ☐ Absent ☐
4. Loss of appetite and weight loss : Present ☐ Absent ☐
5. Haematmesis : Present ☐ Absent ☐
6. Melena(tarry, foul-smelling faces due to oxidized iron from Haemoglobin) : Present ☐ Absent ☐
7. Fatigue : Present ☐ Absent ☐
8. Heartburn : Present ☐ Absent ☐
9. Hunger : Present ☐ Absent ☐

**CLINICAL EVALUATION:**

1. Cardio vascular system : Normal ☐ Abnormal ☐ Details _____
2. Respiratory system : Normal ☐ Abnormal ☐ Details _____
3. Central nervous system : Normal ☐ Abnormal ☐ Details _____
4. Urogenital system : Normal ☐ Abnormal ☐ Details _____

**SIDDHA PARAMETERS:**

1. Naa : Normal ☐ Abnormal ☐ Details _____
2. Niram : Normal ☐ Abnormal ☐ Details _____
3. Mozhi : Normal ☐ Abnormal ☐ Details _____
4. Vizhi : Normal ☐ Abnormal ☐ Details _____
5. Malam : Normal ☐ Abnormal ☐ Details _____
6. Moothiram : Normal ☐ Abnormal ☐ Details _____
7. Sparisam : Normal ☐ Abnormal ☐ Details _____
8. Naadi : Normal ☐ Abnormal ☐ Details _____

**RESULT:**

1. GOOD : ☐
2. FAIR : ☐
3. POOR : ☐
4. NO RESPONSE : ☐

**SIGNATURE OF MEDICAL OFFICER** **SIGNATURE OF INVESTIGATOR**

**LABORATORY INVESTIGATION AND CLINICAL PARAMETERS**

1. Centre : _____
2. Code no : _____ Level of study: OPD/IPD
3. Name of the patient : _____
4. Age : : sex: Male ☐ Female ☐

5. Date and month of assessment : _____

**CLINICAL PARAMETERS:**

**Urine:** Albumin, Sugar, Deposits.

**MOTION;** Ova ,Cyst

**HAEMOGRAM** :Total WBC Count, RBC, Hb, ESR, Barium meal,Endoscopy,Ultra sound

**DEPARTMENT OF POST GRATUATE GUNAPADAM BRANCH,**

**GOVERNMENT SIDDHA MEDICAL COLLEGE,**

**CHENNAI-106.**

**ARIGNAR ANNA GOVERNMENT HOSPITAL FOR**

**INDIAN MEDICINE AND HOMEOPATHY, ARUMBAKKAM,**

**CHENNAI-106.**

**OPEN CLINICAL TRAIL PHASE II B**

**HEPATOPROTECTIVE ACTIVITY OF**

***"ARITHIRAADHI CHOORANAM"***

**FORM II**

16. Centre :
17. Code no : Level of study: OPD/IPD
18. Name of the patient :
19. Address :

20. Age : : sex: Male ☐ Female ☐
21. Educational Status :
22. Occupation :
23. Income :

24. Religion : H ☐ M ☐ CH ☐ S ☐
25. Marital Status :

11. Date of Admission :

12. Date of Discharge :

13. Diagnosis :

**PERSONAL HISTORY:**

26. Food habits : Veg ☐ Non Veg ☐ Veg/Egg ☐
27. Addiction : None ☐ Smoking ☐ Snuff ☐
Ganja ☐ Alcohol ☐ Opium ☐
28. Sleep : Good ☐ Distributed ☐ Insomnia ☐
29. Presence of anxiety : Yes ☐ No ☐
30. Naadi : Vatham ☐ Pitham ☐ Kapam ☐ Thontham ☐

**FAMILY HISTORY:**

6. Hypertension : Yes ☐ NO ☐
7. Diabetes mellitus : Yes ☐ NO ☐
8. Tuberculosis : Yes ☐ NO ☐
9. IHD/MI/MS/AS : Yes ☐ NO ☐
10. If any other disease specify : Yes ☐ NO ☐

**PRESENTING SYMPTOMS:**

| | Yes | No | Duration (WEEKS) |
|---|---|---|---|
| 14. Anorexia : | ☐ | ☐ | _____ |
| 15. Nausea : | ☐ | ☐ | _____ |
| 16. Vomiting : | ☐ | ☐ | _____ |
| 17. Fatigue : | ☐ | ☐ | _____ |
| 18. Insomnia : | ☐ | ☐ | _____ |
| 19. Yellow colored eyes : | ☐ | ☐ | _____ |
| 20. Yellow colored urine : | ☐ | ☐ | _____ |
| 21. Pale coloured motion : | ☐ | ☐ | _____ |
| 22. Skin itching : | ☐ | ☐ | _____ |
| 23. Pain abdomen : | ☐ | ☐ | _____ |
| 24. Fever : | ☐ | ☐ | _____ |
| 25. Diarrhoea : | ☐ | ☐ | _____ |
| 26. Hematemesis : | ☐ | ☐ | _____ |
| 27. If any other disease : | _____ | | |

**HISTORY OF PRESENT ILLNESS:**

☐

Onset of Jaundice : Acute ☐ Chronic

**PAST HISTORY:**

18. Date of diagnosis : _____

19. Duration of disease : _____ months/years
20. Source of HBV infection : _____
21. High risk Group : Medical worker Paramedical worker
Sex worker others
Non – High risk group
22. Alcohol : Yes ☐ No ☐
23. Cancer(other organs) : Yes ☐ No ☐
24. Sedentary life style : Yes ☐ No ☐
25. Achlorhydria : Yes ☐ No ☐
26. Tuberculosis : Yes ☐ No ☐
27. Diabetes : Yes ☐ No ☐
28. Nephritis : Yes ☐ No ☐
29. Syphilis : Yes ☐ No ☐
30. AIDS : Yes ☐ No ☐
31. Pt.underwent any surgery : Yes ☐ No ☐
32. UTI : Yes ☐ No ☐
33. Others : If Yes , Specify _____

**HISTORY OF PREVIOUS TREATMENT:** Yes No ☐ ☐

**If yes, give details as follows:**

**PHYSICAL EXAMINATION:**

10. Height (cm) : _____
11. Weight (cm) : _____
12. Pulse : _____
13. Blood pressure : _____
14. Temperature : _____
15. RR : _____ /minute
16. Anaemia : Present ☐ Absent ☐
17. Lymphadenopathy : Present ☐ Absent ☐
18. Pigmentation : Present ☐ Absent ☐

**PRESENTING SIGNS:**

10. Odema feet : Present ☐ Absent ☐
11. Icterus : Presen ☐ Absent ☐
12. Spider : Present ☐ Absent ☐
13. Gynacomastia : Present ☐ Absent ☐
14. Liver :Palpable ☐ Not palpable ☐
15. Splenomegaly : Present ☐ Absent ☐
16. Ascites : Present ☐ Absent ☐
17. Encephalopathy : Present ☐ Absent ☐

**CLINICAL EVALUATION:**

5. Cardio vascular system : Normal ☐ Abnormal ☐ Details _____
6. Respiratory system : Normal ☐ Abnormal ☐ Details _____
7. Central nervous system : Normal ☐ Abnormal ☐ Details _____
8. Urogenital system : Normal ☐ Abnormal ☐ Details _____

**RESULT:**

5. GOOD : ☐
6. FAIR : ☐

7. POOR : ☐
8. NO RESPONSE : ☐

**SIGNATURE OF MEDICAL OFFICER SIGNATURE OF INVESTIGATOR**

**LABORATORY INVESTIGATION AND CLINICAL PARAMETERS**

5. Centre : _____
6. Code no : _____
   Level of study : OPD/IPD
7. Name of the patient : _____
8. Age : : sex: Male ☐ Female ☐

5. Date and month of assessment : _____

**CLINICAL PARAMETERS:**

Urine: Albumin, Sugar, Deposits,Bile pigments, Bile Salts

**Urine:** Albumin, Sugar, Deposits.

**MOTION;** Ova ,Cyst

**HAEMOGRAM** :Total WBC Count, RBC, Hb, ESR,

Liver function test:

AST,ALT,ALP, Bilirubine, total protein, Albumin., Prothrombin time

# VEL'S COLLEGE OF PHARMACY

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| S.No | Title of The Project | Name of The Investigator | Approval status/Remarks | Project Reference |
|---|---|---|---|---|
| 4. | Evaluation of ovulation inducing activity for infertility and toxicological studies for Uppu parpam. | Dr. N. Kavitha | According to the protocol 36 rats were proposed, but while scrutinizing for pooling the final data, only 30 rats were sanctioned. | XIII/VELS/PCOL/ 04/2000/CPCSEA/I AEC/11.08.2012 |
| 5. | Hepatoprotective activity of Charaparpam by CCL4 induced method in rats | Dr. S. Umera | Total number of animals proposed was 60 rats. But 60 mice were sanctioned because, it was advised to share the control and standard group results. Since the similar pattern of the study has been planned in the same department, hence these data will serve as common. | XIII/VELS/PCOL/ 05/2000/CPCSEA/I AEC/11.08.2012 |
| 6. | A study on Poovarampattai kudineer choornam for the treatment of Swethakuttam. | Dr. A. Chinnaswamy | Total number of animals proposed was 42 rats. But only 36 animals were sanctioned. | XIII/VELS/PCOL/ 06/2000/CPCSEA/I AEC/11.08.2012 |
| 7. | A study of Kanthaga parpam for the treatment of kumbavatham. | Dr. G. Krishnaprakash | Total number of animals proposed was 36 rats and sanctioned. | XIII/VELS/PCOL/ 07/2000/CPCSEA/I AEC/11.08.2012 |
| 8. | Hypolipidemic activity of Kadukkai chooranam. | Dr. F. Priya | Total number of animals proposed was 48 rats, and it was advised to minimize the number to 40 rats only. | XIII/VELS/PCOL/ 08/2000/CPCSEA/I AEC/11.08.2012 |


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| S.No | Title of The Project | Name of The Investigator | Approval status/Remarks | Project Reference |
|---|---|---|---|---|
| 9. | A biochemical study on the effect of Naringenin on cerulean induced toxicity in rats. | Dr. C. N. Deepa | Total number of animals proposed was 36 rats were sanctioned and suggested to reuse the animals sanctioned for safety study after recovery. | XIII/VELS/PCOL/ 09/2000/CPCSEA/I AEC/08.08.12 |
| 10. | Antiulcer activity of Milagathai Choornam. | Dr. F. Priya | 46 rats were proposed for this study, advised to use the common data for standard and control group and the 40 rats were sanctioned. | XIII/VELS/PCOL/ 10/2000/CPCSEA/I AEC/11.08.2012 |
| 11. | Anti ulcer activity and toxicological study of Panai poo (Borassusflabellifer) Choornam. | Dr .P. Kavitha | 46 rats were proposed for this study, advised to use the common data for standard and control group and the 40 rats were sanctioned. | XIII/VELS/PCOL/ 11/2000/CPCSEA/I AEC/11.08.2012 |
| 12. | A study on Arithiraadhi chooranam for hepatoprotective activity. | Dr. P. Kavitha | 46 rats were proposed for this study, advised to use the common data for standard and control group and the 40 rats were sanctioned. | XIII/VELS/PCOL/ 12/2000/CPCSEA/I AEC/11.08.2012 |
| 13. | A study on styptic activity for Menorrhoea and toxicological studies for Sirupeelai. | Dr. N. Kavitha | Total number of animals proposed was 40 mice but 35 mice were sanctioned | XIII/VELS/PCOL/ 13/2000/CPCSEA/I AEC/11.08.2012 |


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